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H. Preston Hoskins, Secretary-Editor, 537 Book Building, Detroit, Mich.

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IN FAVOR OF THE HORSE

In a recent letter to members of the Horse Association of America, Secretary Wayne Dinsmore made a rather significant statement, as follows:

> A year ago, a good farm chunk would buy one good cow, six fat hogs, or seventeen fat lambs; today, the same horse will buy two cows, twelve hogs or twenty-six fat lambs.

This shift in the relative values of farm animals, in favor of the horse, is in line with predictions that have been made, from time to time, by Mr. Dinsmore and others who have been watching the situation closely. Every veterinarian practicing in an agricultural district could give the names of a number of farmers who would like to trade some piece of motor-driven machinery for some good horse flesh. The number of farmers who will be obliged to use horses during the next few years is quite likely to increase rather than decrease. Veterinarians who are in a position to do so should lose no opportunity of bringing to the attention of their clients the results of investigations conducted and reported by the Horse Association of America, from time to time.

Latest estimates of the Department of Agriculture would indicate a shrinkage of almost fifty per cent in the total value of all farm animals during the past two years.

A PRACTITIONERS' NUMBER

This issue of the JOURNAL could very appropriately be called a general practitioners' number. The field of practice is certainly well covered in the eleven papers presented this month. Ten of these were presented at the Kansas City convention. Not only are the papers being published in full, but the discussions which followed these ten papers.

Two of the papers are from the Section on Small Animals and cover two of the most important subjects in canine practice—feeding and fractures. Then we have three papers on two cattle diseases—shipping fever and Bang's disease. There are two papers on poultry diseases—fowl-pox and bacillary white diarrhea. Then we present one paper each on hog cholera, equine parasites, sheep diseases, and parasites of foxes. Following the papers are three very interesting case reports.

It is to be hoped that practitioners will enjoy reading these splendid papers and discussions. Several of the papers were written by practitioners. No less than five were prepared especially for presentation to practitioners and all contain information that the man in practice should have, if he wants to keep abreast of the latest developments and progress in the broad field of veterinary medicine.

WISCONSIN AND OHIO GIVEN CLEAN SLATE

Two more states—Wisconsin and Ohio—have been added to the growing list of modified tuberculosis-free areas, bringing the roll of states in this exalted classification up to six. North Carolina, Maine, Michigan and Indiana preceded Wisconsin and Ohio in the order named. The work in these two states was completed in December, 1931, and on account of the importance of the cattle and dairy industries in these states, the achievement is particularly significant.

The work in the Buckeye and Badger states was started in 1918 but did not gain much momentum until about 1924. A fresh impetus was given to the tuberculosis eradication movement in 1926, when several of the large cities in the Midwest adopted ordinances requiring that all milk sold in these cities be from tuberculosis-free cows. The work was accelerated in order that the dairymen in these states would not be deprived of a market for their milk.

The U. S. Bureau of Animal Industry reports that 10,500,000 tests were made in Wisconsin, with the disclosure of 175,000 reactors (1.66 per cent). In Ohio, about 4,000,000 cattle were tested and approximately 90,000 reactors (2.25 per cent) found.

During the campaign in Wisconsin, there was practically no serious opposition at any time. In Ohio, trouble cropped out on several occasions but the veterinary officials were sustained by the higher courts in each case, much to the discomfiture of the paid propagandists who were largely responsible for the opposition to the test.

The economic advantages of freedom from tuberculosis are quite apparent in both Wisconsin and Ohio. A large part of the milk from the herds in these states is sold in the large cities, where there is a demand for milk from clean cows. In addition, both states sell many surplus cattle to other states, particularly in the East. Wisconsin is said to ship more dairy cows interstate than any other state in the Union. It is perfectly natural that buyers should go to those states where there is the least risk involved, as far as tuberculosis is concerned.

As time goes on, those states which have been backward in eradicating tuberculosis will more and more appreciate the advantages which are bound to go with healthy cattle, as they see neighboring states, that are clean, "getting the business." This applies not only to tuberculosis, but to other diseases as well, with particular reference to Bang's disease, which is really the big problem confronting the live stock industry today. In some respects Bang's disease is like tuberculosis, but it has enough points of difference to make an entirely different plan of eradication appear necessary.

CHANGES OF ADDRESS

The editorial published in the December issue of the JOURNAL, relative to the new edition of the A. V. M. A. Membership Directory, in connection with which a request was made for members to report changes of address, brought a response entirely beyond our expectations. During the months of December and January approximately 200 changes of address were brought to our notice in one way or another.

Many of the changes reported were trifling, such as a change of box number or street number, but nevertheless, every change, no matter how slight, involves six operations as far as our office

records are concerned. From this fact it may be seen that some 1200 operations were involved in bringing our records up to date. This unexpectedly large number of responses to our request has delayed the work on the Directory to some extent, but we expect to have it ready for distribution some time this month.

While on the subject of changes of address, we wish to direct attention to something that happens all too frequently, and that is the giving of different addresses each year when the annual dues are remitted. We refer to those cases where there has been no actual change of address, but, through carelessness or something else, the member gives an address different from the one on our books. Here is an exaggerated example, a case of a member giving five different addresses on as many different occasions, when actually there was no change:

(1927)	Sixth and Broad Streets	(His office)
(1928)	600 Broad Street	(Also his office)
(1929)	819 West Sixth Street	(His residence)
(1930)	Box 411	(His post office box)
(1931)	600 North Broad Street	(His office again)

Just keep in mind that we have no accurate way of telling whether there is an actual change of address or whether the old one is still good. Our policy has always been to consider it a change, if the address given is different from the one already on our books, unless advised to the contrary.

Through the kindness of several members we have been able to locate three of the lost members whose names were published last month. In the meantime we have lost several more, so a revised list is presented this month, in the hope that we will be able to locate some or all of the lost members. They are:

Childs, J. W., c/o Dr. J. E. Van Sant, 523 E. 19th St., Bakersfield, Calif.
Denney, Paul O., 42 W. 43rd St., New York, N. Y.
Harrison, W. E., 155 S. Ventura Ave., Ventura, Calif.
Miller, W. C., 459 W. 43rd St., New York, N. Y.
Murphy, G. H., 1308 W. 68th St., Chicago, Ill.
Schmidt, A. I., 1206 City Park, Houston, Texas.
Steckel, Leo M., Rm. 2302, 1350 Broadway, New York, N. Y.
Whealy, J. A., 1404 W. 9th St., Sioux Falls, S. Dak.
Whitmore, Wm. W., Box 244, Creston, Iowa.
Young, John O., 626 Clay St., Topeka, Kans.

According to a recent publication of the U. S. Public Health Service, undulant fever is a reportable disease in 32 states and not reportable in 7. Information was lacking for the nine other states.

APPLICATIONS FOR MEMBERSHIP

(See January, 1932, JOURNAL)

FIRST LISTING

BARCLAY, ELDON C. R. 1, Box 61, Romeo, Mich.
D. V. M., Michigan State College, 1929
Vouchers: E. T. Hallman and B. J. Killham.

BIRCHER, IRWIN G. 29 Meridith St., Rochester, N. Y.
D. V. M., Cornell University, 1929
Vouchers: C. E. Hayden and M. G. Fincher.

DRAKE, E. H. Leesburg, Va.
D. V. S., United States College of Veterinary Surgeons, 1907
Vouchers: A. J. Sipos and I. D. Wilson.

GRACE, REGINALD L. 90 Elm St., Potsdam, N. Y.
D. V. M., Cornell University, 1927
Vouchers: Don A. Boardman and C. E. Hayden.

GRAVES, PAGE M. Culpeper, Va.
D. V. S., United States College of Veterinary Surgeons, 1912
Vouchers: A. J. Sipos and I. D. Wilson.

GRUBB, W. H. Purcellville, Va.
D. V. M., George Washington University, 1916
Vouchers: A. J. Sipos and I. D. Wilson.

HESS, ORLANDO B. Fostoria, Ohio
D. V. S., National Veterinary College, 1894
Vouchers: H. D. Sheeran and R. Thumann.

MCCREARY, VIRGIL D. Box 73, Brewton, Ala.
D. V. M., Alabama Polytechnic Institute, 1931
Vouchers: C. A. Cary and M. W. Emmel.

OLSON, CARL, JR. Mayo Foundation, Rochester, Minn.
D. V. M., Iowa State College, 1931
Vouchers: Wm. H. Feldman and C. F. Schlotthauer.

SUDDATH, ROBERT O. Box 244, McRae, Ga.
D. V. M., Alabama Polytechnic Institute, 1919
Vouchers: L. A. Mosher and C. A. Cary.

TORREY, JAMES P. University of Illinois, Urbana, Ill.
M. S., Mississippi A. & M. College, 1924
D. V. M., Michigan State College, 1931
Vouchers: Robert Graham and Frank Thorp, Jr.

Applications Pending

SECOND LISTING

(See January, 1932, JOURNAL)

Buchanan, W. S., Box 94, Tinley Park, Ill.
Heinsen, Edward C., 422 Madison St., Port Clinton, Ohio.
Joneschild, Edward M., Livestock Sanitary Board, Helena, Mont.
Panisset, Maurice, Oka, Quebec.
Pritchard, Robert M., Seabright, Santa Cruz, Calif.

The amount which should accompany an application filed this month is $9.58, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

The number of new applications listed this month is 37.5 per cent more than for the corresponding month of 1931. This is leap year and February has an extra day. Why not use part of it to get a new member?

COMING VETERINARY MEETINGS

Alabama Veterinary Medical Association and Short Course for Practitioners. Alabama Polytechnic Institute, Auburn, Ala. February 1-6, 1932. Dr. C. A. Cary, Secretary, Auburn, Ala.

Connecticut Veterinary Medical Association. Hotel Garde, Hartford, Conn. February 3, 1932. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. February 3, 1932. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill., February 9, 1932. Dr. E. E. Sweebe, Secretary, 14th St. & Sheridan Rd., North Chicago, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. February 9, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. February 10, 1932. Dr. H. Preston Hoskins, Secretary, 537 Book Bldg., Detroit, Mich.

Louisiana Veterinary Medical Association. Dalrymple Hall, Louisiana State University, Baton Rouge, La. February 10-11, 1932. Dr. H. A. Burton, Secretary, Alexandria, La.

Kansas Veterinary Medical Association and Kansas State College Conference for Veterinarians. Manhattan, Kans. February 10-11, 1932. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans.

Hudson Valley Veterinary Medical Society. Albany, N. Y. February 10, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Ontario Veterinary Association. Ontario Research Foundation, Queens Park, Toronto, Ont. February 11, 1932. Dr. H. M. LeGard, Secretary, 335 N. Main St., Weston, Ont.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. February 17, 1932. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.

Ohio State University, Conference for Veterinarians at. Ohio State University, Columbus, Ohio. March 23-25, 1932. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

THE TREATMENT OF FRACTURES*

By E. B. DIBBELL, *Baltimore, Md.*

There is no serious work published in veterinary literature to guide the veterinarian in the handling of fractures and dislocations in small animals. The increase in auto traffic in the past fifteen years, with the resultant injuries to our pets, has made the need of an interchange of ideas, methods and procedures between practitioners advantageous. Not alone is the city veterinarian interested but the country practitioner also. No longer is the farm pet secure; the country highways are even more dangerous to animal life than the city streets, for here speed is unleashed.

It is not within the scope of this paper to discuss specific fractures. There are basic principles involved and a background of knowledge is needed before one should attempt much fracture work. Some knowledge of the repair of bone, anatomical peculiarities of the different bones and their muscle attachments, and a working idea of traction and fixation form a foundation on which to proceed.

I am free to admit that I have gone to the text-book of the M. D. for some of the following material but have used only that portion of the text that I have found by experience to be practical and have modified some of the suggestion of the M. D., making them by that modification adaptable to fracture work in animals.

FIRST AID

When the animal is first seen, treatment of the existing shock should be our first consideration. Too much unnecessary manipulation is usually done and not enough attention paid to the animal's general condition. A temporary splint should always be applied in "out cases" before their removal to the hospital and also those cases presented at the hospital when, for any reason, a permanent fixation cannot be applied for a day or two. The sooner a fracture is reduced and immobilized, the less will be the swelling, the easier will the patient rest and the better will be your results. There is a prevalent idea of waiting until the "swelling has gone down." This ancient phrase rolls off the tongue easily and sounds important but is unsound. Traumatic reaction is going on all the time, as long as the bones are movable or are out

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

of place. If we can obtain immobilization and reduction soon enough, there will be no swelling to speak of.

Examination

Merely to know that a fracture exists is not sufficient. Note the injury to the soft parts as well as of the bone, so as to visualize properly the problem of obtaining and maintaining reduction as well as the problem of repair and its duration.

In making an examination, begin with painless measures first. Note angulation. Take measurements for length, by comparison with the sound limb, in long-bone fractures. Do not test for crepitus and abnormal motility unless necessary. Too much manipulation causes additional injuries. Your examination will embrace observation for:

1. Deformity.
2. Abnormal mobility.
3. Pain and loss of function.
4. Crepitus.
5. X-ray.

Deformity: Includes changes in the relation of the bone and the shape of the limb. The two limbs must always be compared. It must be known that there has been no previous injury to cause the deformity.

Abnormal mobility: A sign on which too much stress is usually placed. The sign certainly indicates fracture but its absence tells you nothing. In impacted fractures, in fractures of the carpus and tarsus, and in epiphyseal fractures there is no abnormal mobility. In the case of breaks near joints it may be impossible to determine whether the movement is in the joint or near it.

Pain and loss of function: These go together, as the pain is often responsible for the loss of function. Both are present to a degree in all fractures. The amount of pain varies with the location but is nearly always aggravated by movement and pressure.

Crepitus: The grating produced by the friction of the broken ends of the bone or bones. Crepitus is pathognomonic but should not be sought for too vigorously.

X-ray: There are very few veterinary locations today where an x-ray is not available for use. All human hospitals, and a great many physicians have machines. Even the dentist's unit can be used for small-animal work. The value of the x-ray is too well known to enter into a discussion of its merits here.

Treatment

The treatment implies a reposition and an immobilization so that the bones may unite in their normal relations. It is important that there be no deformity and there should also be restoration of function. An example of correction of deformity but lack of return to function is seen in fractures of the humerus, where the bone may repair perfectly but function never returns, due to injury to the musculo-spiral nerve. This injury to the nerve may be due to the same force that caused the fracture, but too often it is due to too tight wrapping of the bandage over the fractured area with injury to the nerve.

In making reduction, violence should be avoided. Gentle and persistent effort is always better than rude haste in overcoming the resistance of muscles and ligaments.

Traction is the chief factor in the reduction of most fractures. In making traction it will be found that, where much overlapping occurs, it can best be done when the animal is under a general anesthetic.

Traction may be immediate and forcible under general anesthesia or steady and continuous, such as the traction employed by the use of the Thomas splint or a modification of that idea. Continuous traction by weights, while possible, taxes the ingenuity of the operator and is unnecessary except in rare cases.

There are some fractures where ordinary surface traction will not effect a reduction. In lower-third femoral breaks the action of the leg muscles pulls the bones so out of line that no amount of surface traction will bring about reduction. Here skeletal traction must be resorted to. Skeletal traction means direct traction to the bone itself by the use of pins or tongs either passed through the bone end or imbedded into the bone surface deep enough to permit direct pulling. The procedure is common in femoral fractures of the human and, while requiring some slight ability on the part of the operator, is just as practical in its use on animals.

Fixation

A decision should be reached in each case of the particular problem presented and the selection of apparatus and material made accordingly. If an anesthetic is deemed necessary to complete a diagnosis or reduce a fracture, everything should be prepared and all material assembled so that the one anesthetic is all that is necessary for the fixation or casting.

Rapidity of repair will depend on the blood supply to the fragments. The head of the femur when detached at the surgical neck is usually very slow to repair and often never does form a bony union because the same injury that caused the fracture disturbed circulation to the proximal fragment. These factors should be taken into consideration when determining the duration of fixation.

The value of any apparatus or material is of less importance than the skill with which it is used. In the very nature of things there can be no unalterable technic employed in gaining fixation. Ultimate functional results will depend on the correction of the displacement and adequate fixation. As I said before, it is not the material used in the fashioning of the splint, nor the type of splint used that determines the outcome. It is the man behind the splint that counts.

Compound Fractures

There are two classes of compound fractures.

First, we have the type in which it is perfectly evident that the wound of the soft parts is made by the protrusion of bone from within, through the skin, with little surface bruising or abrasion.

Second, the injury to the skin and soft tissues where it is evident that trauma from the outside has penetrated to the bone within.

Type 1 may be regarded as a non-infective wound and, after being treated with iodin or other antiseptic, it can be handled as a closed fracture. A window should be left in the bandage over the site of the wound, through which to watch for a possible flare-up, however.

Type 2 should always be regarded as an infected wound and should be handled in somewhat the following manner. With sterile gauze protect the wound from further contamination while the surrounding skin is being cleansed. The skin is painted with tincture of iodin up to the edges of the wound preparatory to operation. The operation called for here demands the excision *en bloc* of traumatized and infected tissues, taking in a generous margin of skin from the wound edges. All dead and dying fat, fascia and muscle are cut away until fresh bleeding occurs. Small bone fragments are removed unless firmly attached to the periosteum. Frequent sterilizing of hands and instruments to

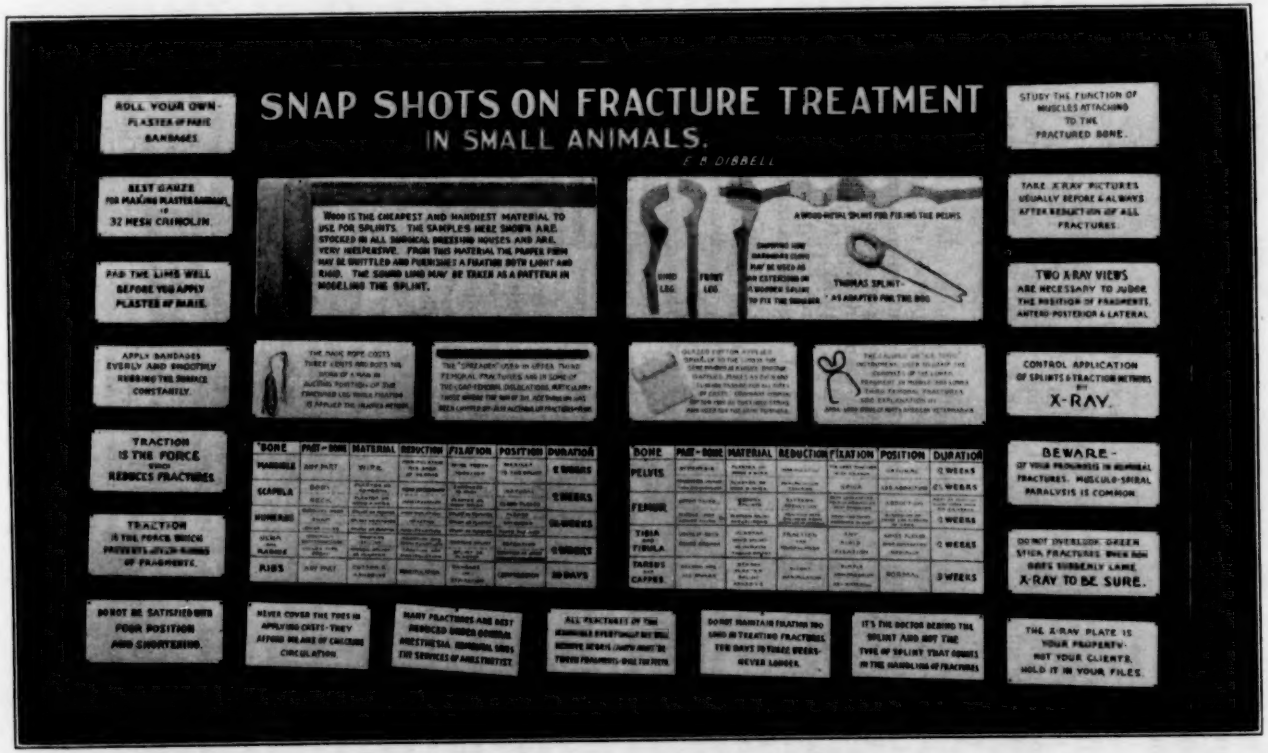

FIG. 1. Exhibit entitled, "Snap Shots on Fracture Treatment in Small Animals."

keep from carrying infection into the deep portions of the wound should be carried out. Drains should be inserted. A window should always be formed in the cast to permit dressing of the wound.

Discussion

Dr. J. V. Lacroix: Dr. Dibbell has given us a splendid, brief discussion of the essentials in his consideration of basic principles that apply in the handling of fractures in dogs. I do not feel prepared to do justice to a discussion of this subject. However, if you will bear with me, I shall mention a few things that have seemed important to me and that have occurred to me as I listened to Dr. Dibbell.

I did not have time to finish reading the notes and exhibits on the board. I must, perforce, refrain from taking up any portion of this in serial order.

I think the caution mentioned in regard to undue manipulation of fractured members in dogs is a timely and wise piece of advice. Fractures that are perfectly obvious do not require manipulation, and that is the case in the majority of instances in our practice, I think. Where particles of bone exist, perforation or puncture of soft structures inevitably is occasioned by manipulation. The process is painful, it adds to traumatism, and tends to no good result.

In general, and without any classification on a pathological basis, we have two kinds of fracture to treat: those which are obvious and those which are occult or vague in character. In the latter class of cases we must rely upon the x-ray or fluoroscope. Of the two, I prefer the radiograph. The fluoroscope is of no use in the pelvic region of dogs of any considerable size. A picture must be taken in such cases. When the x-ray is used and the picture developed, we have something on record.

I think I saved myself at least $500 in attorney fees, time consumed, and so forth, in one instance, by this means alone in handling a fracture of the femur in a show Chow bitch. I took a picture of the fracture at the time it occurred and another after complete recovery had resulted, and proved by means of these views, which included the pelvis, that this subject's usefulness was not impaired for breeding purposes and that her ultimate value was not lessened.

Dr. Dibbell has done considerable work in this field. He is very much more competent to advise you regarding that phase of it which has to do with traction, fixation, the use of the clipper or tong, and many other phases of handling fractures, than I.

I shall conclude by referring briefly to those difficult fractures in dogs, those which I think cause more trouble than any other kind—fractures of the femur in dogs. At least, that has been true in our practice.

Because of the contour of the hip, the morphology, the application of the ordinary splint is precluded. Where there is over-riding, a shortening of the bone, or where there are multiple fractures, the case is indeed difficult.

I think as a class in practice we are too hesitant to explain to the owners of these animals the serious difficulties that are to be encountered in the handling of such cases. With a good x-ray picture that reveals to the owner the conditions with a fair degree of definiteness and a little explanation, we can have a sympathetic client to deal with and pave the way for a better understanding should recovery be only partial or not at all.

Member: What should be done in the case of a dog that has a crooked front leg, so that it is impossible to put on a cast to keep it straight?

Dr. Dibbell: In that case, apply over-correction. In the first place, if you get proper reduction and set, you will probably have a straight limb. If you find you cannot do that and there is some overlapping, if you will over-correct it and check that by x-ray, you can probably get a good line-up; at least, one that nobody else would notice.

Over-correction merely means, if there is a tendency to buckle in one direction, over-correct it in the other direction to compensate for that tendency.

In that particular type of fracture, sometimes we find reduction is very much easier if the wrist is flexed. Sometimes the flexor muscles involved are relieved and the leg put up in a flexed condition.

DR. JOHN F. MCKENNA: Has a schedule been worked out as to how long the splint should be left on, in various fractures, according to the age of the animal?

DR. DIBBELL: We usually check the rate of repair by the x-ray.

DR. HAMLET MOORE: Regarding this matter of fracture of the radius and ulna—I have had much experience with greyhounds. I have been official veterinarian to the greyhound racing tracks in New Orleans ever since that sport was started. I have had as a minimum number thirty-five racing greyhounds with fractured radius and ulna, occurring principally in jumping dogs.

I have never had any trouble with a dog that I set myself. I took cognizance of the advice given by the author of this paper as to the matter of manipulation, but the first thing I did was to give the dog two grains of morphin. I would let him go into the hospital and by the time he got there he was about ready to lie there and say, "Go as far as you like."

I simply put the bones in apposition, then put on a big dry cotton compress and a splint that is not made to shape anything. It is just an ordinary splint which I split into about inch widths and put on four splints. Then I start bandaging. I put on liquid glass—and I know there are going to be a great many questions about liquid glass—and put the cast on. Put the dog to sleep and never bother with him at all. In nine days I would take the cast off, and I have never set a leg on one of those thirty-five dogs that did not go back to racing and perform and win.

I have had any number of dogs sent to New Orleans from Galveston, Miami and Chicago, that had had their legs set and why they were bent, I don't know. In preference to rebreaking the leg, the thing I did was to cut down to the periosteum, dissect and put it back, then with a bone chisel, chisel it out to shape. I simply split the periosteum, turn it back, and with a bone chisel or gouge, gouge out the part that was bent, then put back the periosteum and suture, later taking out the suture. The dogs have always made perfect recoveries.

A dog named "Coon Can," worth $5,000, was brought to me. We all have some one who loves us better than any one else. The owner of this dog was a particular admirer of mine, so when the dog's leg was broken in the last race at eleven o'clock one night, he took the dog to some one else to have its leg set. At about twelve o'clock, after setting the leg, this man told the owner there was only one thing to do and that was to amputate the leg.

You can imagine how that pleased the owner. You can readily understand that a jumper worth $5,000 would be much better off with one front leg gone. The owner would not let him amputate and brought the dog to me. He said, Dr. Moore, I owe you an apology."

I said, "If that is all you owe, it is paid." Of course, that wasn't all I said.

He said, "Will you set this dog's leg?" I told him I would.

I gave the dog two grains of morphin. It was a physical impossibility for four men to straighten the leg, the radius and ulna were so overlapped. I had to dissect the tissues, make an incision from inside and outside from both ends of the fracture so that we could bend the leg and get it down. There was no way to separate the two bones, at least none that I knew. The dog being under full anesthesia, it was easy to bend those bones down.

In putting the cast on, I placed a pocket of absorbent cotton with acriflavine solution, then put on the glass cast and the splint. This dog made a complete recovery and went on and jumped and ran afterwards.

With these bent radii and ulnae, I have never seen one that I have set that showed any malformation or a tendency toward curvature of the bone. I have just chiseled off the curvature and straightened it that way.

DR. T. H. FERGUSON: Years ago, before we found a better method for making a thorough examination of the male bovine, it was often necessary to do a subcutaneous myotomy.

I was wondering, in cases of fractures, where it is next to impossible to keep the bones in apposition, if a subcutaneous myotomy might be used, or

whether it would be indicated in some of the cases where the bone grows crooked or bends due to muscular contraction.

DR. LACROIX: The one place where that is indicated is in fracture of the os calcis. The older men know what I mean. That is the place where subcutaneous myotomy of the Achilles tendon would be an ideal measure, but please remember to do that operation rather high up and be sure that you find the ends in order to join them later; else you will get that exaggerated flexion ever afterward.

There is something to open reduction treatment, in the ordinary sense, and in many instances, to subcutaneous myotomies.

CHAIRMAN PARKER: You all, no doubt, have plenty of instances of pseudo-luxation in fracture of the radius and ulna. Why not use this opportunity to ask Dr. Dibbell how to prevent it? We have all encountered it, if we have set many legs. I have not had many but enough to teach me one thing, and that is, the dog must be more quiet than with any other fracture I know of when he has a fracture of both radius and ulna. The pseudo-luxation or false joint that sometimes results is caused by too much exercising of the dog, or possibly too loose a bandage.

DR. DIBBELL: I think if there is proper metabolism, that is a point to be considered in the repair of fractures as well as the type of bandage. When you have a puppy that is rickety, as fast as you apply and take off the bandage you will find new fractured areas. I have often seen cases where I am sure it was nothing but improper metabolism, lack of calcium, having much to do with the condition, and whether it is real or imaginary, I feel that calcium and parathyroid have helped me in the repair of bone.

We also use the violet rays or Alpine lamp in connection with the calcium in cases where there is this tendency. I think it is hard to say definitely that that is entirely responsible, but my impression is, from my own experience, that it has been of decided benefit.

DR. O. F. REIHART: How did you administer your calcium?

DR. DIBBELL: Simply as calcium lactate, with the parathyroid extract.

DR. REIHART: Do you believe you get better increase of calcium in that way than with the violet ray or Alpine lamp, or vice versa?

DR. DIBBELL: In the first place, I don't pretend to know much about calcium metabolism. Usually I use the lamp in connection with the administration of the calcium and a little parathyroid. What the ratio of virtue is in each of these factors I am not at all competent to say.

DR. REIHART: We found we got better results from the ultraviolet ray lamp or the Alpine lamp than from internal calcium administration because so little of the calcium is taken up when administered orally. We use parathymone, which raises the calcium metabolism very quickly. We find it better than anything else. It is used hyperdermically.

DR. K. U. JONES: How often do you repeat the injection?

DR. REIHART: We use two-cc doses about three days apart.

DR. C. W. BOWER: I was interested in the question of the crooked front leg. I am wondering if that is where I am wrong. Perhaps it is the same as I explained to the boys in Iowa, last winter, about the application of the splint, that is, the long splint underneath the arm.

We have found that this helps. I would not say it was perfect, but before adopting such a splint we had a big percentage of our ulna fractures go crooked. I judge that is what the doctor had in mind when he asked the question. We found in putting on our lateral splints they would either slip or be loose, or else the dog would put its weight on its foot too soon, and we attributed a lot of our trouble to that.

For the past three or four years we have been using a long splint that goes down underneath the surface of the limb after reduction has taken place. It is the size of the average lath and is well padded. We lay the leg right on it and let the splint extend perhaps an inch and a half or two inches below the paw, thereby giving a crutch for the dog to walk on. You have noticed that the dog will start using the leg the next day and if he walks on the foot, the leg will commence to bend.

With this long splint we have virtually made the dog a peg-leg and he will commence pegging on this right away. After nine to twelve days, we take

off the splint. We have found since using that method we have a larger percentage of straighter legs. It is not perfect, but it is a help.

DR. H. W. YOUNG: I think Dr. Bower and Dr. Parker will remember that at the Missouri Valley clinic last winter we had a wire-haired terrier with a broken ulna and radius. The dog was brought in probably three weeks after the break and had not been treated. The radius and ulna were both fractured and Dr. Parker set the leg, which was not solid at the time.

I mean to cast no reflections, but that formed a false joint, about an inch or less below the elbow. Apparently it healed and in about three weeks the dog started to use the leg. I noticed a slight movement of the leg and the people wanted the dog destroyed because they didn't want it that way.

I thought I would experiment a little. I took a small bistoury, and went in and scraped the bone, cutting into the bone. I was not yet hardened, due to lack of calcium, I think. It was still soft on the surface of the false joint. I irritated that. Then I put the dog on calcium lactate and also glucose hypodermically, put the splint back on the limb, and in less than ten days it was as hard as could be. I corrected the false joint in that way.

I wonder if any one here has attempted scarifying a joint, where the ends have united, so to speak, but are still soft, setting up more irritation with a sharp bistoury and then putting the dog on calcium and glucose. This dog now has four sound legs. The leg is slightly curved but it is sound and the dog uses it perfectly.

CHAIRMAN PARKER: I remember that the fracture was well up to thes capulo-humeral articulation. It had been fractured for a number of weeks and had been held crooked. I am glad to learn about that because there was a great possibility of a false joint forming because it had gone so long without having anything done to it.

I have had that experience but not in the last four or five years. I found I was letting my patients have too much freedom. My first case was a large Collie that had had its leg broken about half-way down in the radius and ulna. In spite of everything the owner would do, the dog persisted in going back and forth after the cows. The mischief was done, of course, and that dog is the only dog I can think of that has a permanent false joint. I have had a number that recovered after three or four months. They would spontaneously solidify and be all right, but this one is a living example of a false joint and always will be.

Premise Disinfectants Investigated

The United States Food and Drug Administration is conducting a survey of the "premise disinfectants" now on the market, according to Dr. H. E. Moskey. Several of these preparations, labeled with claims which might create a false sense of security in the mind of the user, have already been found. The Administration holds that label statements implying that a disinfectant or antiseptic can prevent diseases in live stock, under all conditions, are unwarranted and violate federal statutes. Favorite diseases for exploiting these preparations are those that are most generally prevalent among farm animals, including hog cholera, abortion of cattle (Bang's disease) and roup of chickens.

Antiseptics and disinfectants recommended for use in drinking water for poultry and animals are receiving official attention. The fact that the preparation will disinfect water does not prove that it will prevent the spread of a disease if other avenues are left open.

A CONSIDERATION OF THE DIET OF DOGS*

By CARL F. SCHLOTTHAUER, *Rochester, Minnesota*

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Biologically, dogs are grouped with the carnivora. However, like man, they are omnivorous and are commonly maintained on about the same foods as are available for man. Because of this, their diet frequently is controlled by environment. For example, in the Arctic regions, where vegetables and cereals are scarce and many dogs are used for transportation, they must subsist chiefly on fish and the meat of game animals because it is more available; in warmer climates, and especially in large cities in which vegetables and cereals are abundant, dogs frequently are maintained on practically meatless diets. Indeed, many times dogs are forced to subsist on meatless diets because their owners think meat causes worms or makes dogs vicious. Again, there are some persons who think the dog is incapable of digesting starchy foods. Both theories are false. Dogs can subsist on either meat or cereals if properly prepared. However, from the standpoint of health and economy, a mixed diet of meat, vegetables and cereals is recommended.

Anatomically, the dog's digestive system is especially suited to the handling of concentrated food. He has a relatively large stomach and a short intestinal tract. Meat is the most ideal concentrated food, and should be included in most diets. Ashmont[1] stated that a diet containing meat is suitable for most breeds. All authors agree that beef is the best meat. However, the most costly cuts are not required. Mellanby[2] has shown that suet is beneficial to good dental development. Therefore, the most inexpensive cuts of beef are perhaps best, for they generally have a better admixture of lean meat and suet. Spaulding[3] stated that horse meat is equally as good as beef but that it may have a more laxative effect. Veal and mutton, too, are excellent, but they have no advantage over horse meat or beef. Fowl and fish, because of the danger from bones, and because of the cost, are not generally recommended. Meat may be fed raw or cooked. Spaulding expressed the opinion that for general use cooked meat is better than raw meat when fed in large quantities, for

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it is less likely to cause intestinal disorders. Childrey, Alvarez and Mann[4] found that raw meat was more suitable to dogs than was cooked meat. However, cooking may be necessary at times to kill parasites. There is some difference of opinion as regards the value of mincing or grinding meat for dogs. Childrey, Alvarez and Mann, in their recent experiments with colectomized dogs, found that meat when fed in lumps was better digested than when minced. They also found this to be true of some vegetables. This indicates that dogs apparently are doing the correct thing when they bolt their food. All authors agree that liver and pork are undesirable meats for the regular diet of dogs.

The quantity of meat that a dog should receive will depend, of course, on his breed, age, condition and activities. Toy dogs should receive very little meat, whereas working dogs and brood matrons should be given an ample allowance.

Practically all cereal foods are suitable to be added to a dog's ration when well cooked or baked. White bread, well cooked rice, oatmeal, cornmeal and farina all are excellent. Then, too, there are a great number of commercially prepared dog biscuits, puppy-meals and breakfast foods to select from.

Garden vegetables generally are not so digestible as cereals. They should be well cooked and fed in moderate quantity. Asparagus, carrots, potatoes, peas, beans, beets and onions all are suitable. Cabbage in the form of sauerkraut can be added to the above list of vegetables. When cooked with meat it is relished by most dogs.

Sweet milk and buttermilk both are excellent. When combined with cereals their digestibility apparently is enhanced. Corn syrup, when added to milk, greatly increases its nutritive value. This milk and syrup mixture is especially valuable to sustain and build up weak and debilitated animals. Cottage cheese is the only cheese that can be recommended as food for dogs. It is easily digested.

Contrary to general belief, raw eggs are not a good food when fed alone. Childrey, Alvarez and Mann found them to be indigestible. However, when mixed with milk or cereals their digestibility is greatly enhanced and they make an excellent food. Soft boiled eggs are superior to raw eggs when fed without other foods.

Persons who do not wish to prepare special foods for their dogs can now obtain any number of good commercially prepared dog foods. Almost all large kennels find it advantageous to resort to these foods for a part or all of their food requirements.

The condition, breed and activity of the dog are the governing factors as regards how much, what, and when it should be fed. Brood matrons, puppies, working dogs, and those inclined to become obese should receive a protein-rich diet, whereas adult dogs in poor flesh should be given as abundance of carbohydrates. Thus, by careful observation and correct apportioning of the various food products, it should be possible to maintain a dog in the condition desired.

Contrary to general opinion, bones and hard foods are not essential for proper dental and jaw development. Mellanby has shown that the mineral and vitamin constituents of food and not its physical character are more important in this regard. A soft, pulpy diet, if it contains the proper minerals and vitamins, will produce good teeth and jaws.

It is the habit of most dogs to lie down and rest or sleep following a large meal. Therefore, dogs that are inclined to be noisy at night should receive their largest meal in the evening. On the other hand, watch dogs should receive very little food at night. When house-training puppies, it may be advisable to give them their largest meal in the morning, so that they may be put out of doors at frequent intervals during the day. Much soiling of the house can be avoided by so doing.

One large meal is better than a number of frequent small feedings, for each time food is taken into the stomach peristalsis is induced. Frequent feeding causes the food to be forced through the digestive tract at too rapid a rate to permit complete digestion. One or two meals each day are sufficient for adult dogs. Puppies, however, require three or more until they obtain their maximal growth. Caged dogs, in kennels, might well be fasted one day each week. This is practiced in some large zoölogical parks with excellent results.

References

[1]Ashmont: Kennel Secrets: How to Breed, Exhibit and Manage Dogs. (Boston, Little, Brown and Company, 1904), pp. 3-80.

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[3]Spaulding, R. H.: Your Dog and Your Cat: How to care for them. (New York, D. Appleton and Company, 1929), pp. 92-114.

[4]Childrey, J. H., Alvarez, W. C. and Mann, F. C: Digestion: Efficiency with various foods and under various conditions. Arch. Int. Med., xlvi (1930), pp. 361-374.

Discussion

Dr. Schlotthauer: The matter of fasting is, I think, very advisable. We practice it in our kennels and since practicing it we have reduced digestive troubles by a large percentage. We know that when a dog is at large, and especially before it becomes domesticated, it gets only one meal every two or three days.

We give them dog biscuits two days each week as a carbohydrate. Three days each week they get a cooked mash composed of equal parts of ground horse meat—that is, freshly slaughtered horse meat—in which ground bone is included, and oatmeal and cornmeal. That is thoroughly cooked and they receive that three days each week. One day each week they receive raw meat. We find the raw meat is necessary to prevent osteoporosis, or bone trouble. If they continue on cooked diets for long, the bones get in such condition that one could cut through a rib, spine or other bones with a knife. On the seventh day these dogs receive nothing, and they all seem to thrive on this diet I have outlined.

DR. L. W. GOSS: It is very convenient to use the meat scrap that is on the market. I am wondering if that has not been cooked to the extent that it has lost much of its value as a meat food. I have been feeding it for some time in connection with shredded wheat, very largely, and buttermilk, and have had fairly good success. However, I do not feed many vegetables and I think probably I should feed more, and also that I should feed raw meat in connection with this scrap. I have been trying about fifty per cent meat scrap without any blood in it.

DR. SCHLOTTHAUER: I should think the diet would be quite satisfactory for an adult dog, especially. As for feeding vegetables, my paper is largely intended to cover the feeding of the house pet. As I stated, any of these vegetables are all right. When a person comes to you and asks what to feed his dog, you can tell him what Dr. Childrey, who conducted this experiment, said, "Everything."

One of the startling things to me was to learn about the digestibility of potatoes providing that they are fed in lumps. When they are fed in mashed form they pass through as a starch product. If they are fed in lumps they seem to be retained in the stomach long enough to be digested quite well. Rice was found to be the most easily digested of all foods, and white bread next. Starchy things were really very good and left but little residue. These were colectomized dogs, so that the residue could be calculated.

The syrup and milk mixture I mentioned is one we can all employ to advantage. It is very nutritive and is fine for a dog that is unable to retain solid foods or much of anything else. I have seen distemper dogs that would not take a thing built up remarkably if given a small amount of milk and syrup through a stomach-tube. We know that sugar is needed to detoxify the liver. The severe toxic symptoms noted in distemper may be the result of toxic disturbance in the liver. Sugar is a readily available food and can be given intravenously.

Glucose or Karo syrup and milk should be used much more extensively than they are. If the dog will not drink the mixture, it can be given by means of a stomach-tube. We generally feed the stock mixture which we make up; we feed some every day to certain dogs. Our mixture consists of one gallon of syrup for each ten gallons of milk.

DR. H. W. YOUNG: I would like to ask the method of feeding the puppies at weaning time, or a puppy that has lost its mother. Do you make a differentiation in the diet for large and small breeds?

DR. SCHLOTTHAUER: From an investigation of what various people feed their puppies at weaning time, I could say almost anything and be right. I might be feeding something and getting by with it, whereas some one else could not. But in feeding puppies I would stick to rice, white bread, oatmeal and milk. Sometimes they will not drink milk, in which case put it on the food.

Give them at least one-fourth meat and make that raw beef, if they will eat it. As I have said, a dog is probably the most adaptable creature, next to human beings. Dogs will eat everything from raw cereals to meat alone and apparently get along as well as humans. Of course, not all humans should eat everything, either, but many do.

DR. S. W. HAIGLER: It has been my understanding that the internal organs, liver, heart and kidneys, are rich in vitamins and constitute a very good food.

DR. SCHLOTTHAUER: In reading various books you will find that most men say not to feed liver. I doubt if any of them have any experimental backing to show why they think it should not be fed. It is just like raw eggs—we all feed raw eggs without any other food and without having them recommended

as a diet. Raw eggs are totally indigestible when fed alone. One author says boiled eggs are as indigestible as shoe leather. Another author says they are readily digestible if fed with something else. I know some people in Rochester who feed their dogs nothing but liver.

DR. HAMLET MOORE: To excite a discussion—if I were making a book I would lay ten to one that nine men out of ten in this room are of the impression that the dog is not a starch-digesting animal, that two of the prohibitives are Irish potatoes and white bread. I have heard any number of men say that the prohibitives for feeding dogs, in the order named, were Irish potatoes in any form, and white bread untoasted. They do qualify by saying white bread toasted is made digestible because in that way the starch has been broken up. About the same is true of raw eggs although perhaps not quite so emphatically.

I know that racing greyhounds are fed concentrates but the dogs get whole wheat bread when they are laid off. If you go into one of those kennels you will find two tons of it split and nailed on the posts, picking up the germs to flavor it, and sun-bathing to dry it.

I mention this because I am one of those nine-tenths who have made the statement two or three times in my career and have classed those two foods as heading the list of prohibitives, that is, Irish potatoes in any form and white bread untoasted.

You have heard Dr. Glass make the statement any number of times that there is nothing much better than tripe to be fed to dogs. I was being shown around the S. P. C. A. building by Dr. Garbutt in New York recently. They buy tripe in frozen bundles of one hundred pounds and make a mixture of it and if a man were hungry, he could come very near to eating it himself. Fortunately, I had had breakfast before I went there.

DR. SCHLOTTHAUER: I think one reason that we came to the conclusion that starch was bad, is that the dog belongs to the order carnivore. If you feed potatoes or any starch food in mash form, or meat as hamburger, in large enough quantities, it is not readily digestible. However, if fed in chunks, as the dog would eat it in the natural state, it would be digestible.

MEMBER: A fellow was raising police dogs and about all he had to feed them was new potatoes, which were cheap. He cooked them and these puppies had almost an exclusive diet of Irish potatoes. In one or two weeks the puppies commenced to get lame. Finally the whole litter got in such condition they couldn't stand on their feet.

Doctor Moore spoke about the prejudice against Irish potatoes and white bread, and I had heard of it. There was this example right at home.

This young man changed the diet to raw meat entirely. In four or five days the puppies were on their feet and doing fine. Were the potatoes responsible for their condition in the first place? I have always maintained that they were.

DR. SCHLOTTHAUER: The potatoes did not do it but the lack of raw meat did. They should have had raw meat at least once a week, a little every day would be better.

MEMBER: I would like to ask Doctor Schlotthauer if he prescribes any specific diet in non-parasitic skin diseases.

DR. SCHLOTTHAUER: In that respect I have been a fool for luck, as I learned yesterday. I prescribe raw beef in non-parasitic skin conditions which I cannot attribute to any definite cause. My first case was that of a bull terrier owned by a lady in Rochester. He developed an erythema of the skin, and she applied ointment. The condition continued and became worse, so that instead of being a small spot it was all over the dog. She brought him to me for treatment after he was entirely covered with eczema. All I did was to give him a bath and put him on a raw meat diet for two weeks. When she came for him in two weeks the dog had a nice coat started and was a rather nice looking dog.

I have had several cases similar to that since, but you understand, of course, that these dogs are negative for parasites. I had not thought about the anal pouches until Dr. Khuen mentioned that condition yesterday. I could not attribute it to any other thing. It was a moist condition. Four or five dogs that I treated in this way cleared up nicely.

DR. J. C. WRIGHT: I would like to ask Doctor Schlotthauer what diet he prescribes for puppies not over a week old. We often have fine bird dogs in the South that have never been able to raise their puppies; yet their breeding

strains are worth thousands of dollars. We have had laboratory tests run on the milk and apparently there is nothing in it. Yet the puppies will die in three to five to seven days. They will begin to dwindle, perspire freely over the abdomen and then get into a complete perspiration all over. What kind of a diet should be given to puppies like that? I think we have tried about everything.

DR. SCHLOTTHAUER: I haven't had much experience with that, but the few dogs I have seen raised on milk have been raised on milk of about the same mixture they commonly feed babies in most hospitals, which is diluted cow's milk. It is about half milk and half boiled water. After being boiled for two or three minutes, a small amount of syrup or sugar is added. The concentration can be increased as the puppy gets older.

I know of several that have been raised that way, including a Pekingese. They are not remarkably well but they have survived.

DR. WRIGHT: A leading baby specialist of Atlanta prescribed two teaspoonsful of Karo syrup to a glass of water, a teaspoonful of lime water, and two teaspoonsful of Eagle brand milk made up with lukewarm water. He said that is the diet he puts young infants on and so far, we have had better results with that diet than anything we have ever tried but it is not entirely successful.

DR. H. W. YOUNG: I would like to give you gentlemen our way of feeding puppies. We had one brought to the hospital last night that was born Saturday night—a Cesarean, and the mother died. We raise puppies every year that never have an opportunity of nursing. We raise a large percentage of them although we sometimes have deaths, of course.

We start with diluted milk, one-third to one-half water, the first day, using Carnation milk unsweetened. Then we start adding one drop of U. S. P. cod-liver oil. A good grade will contain a lot of vitamins D and A.

As far as lime water is concerned, I believe our latest authorities say that calcium will not be taken up without the presence of vitamin D. We do not give lime water. We use cod-liver oil, and after three or four days, we dilute the Carnation milk with cow's milk, skimmed, and as soon as the puppy will swallow or suck a piece of meat, we give him a piece of raw lean beef with no fat, thereby giving him his vitamin B. Vitamin B is not stored in the body, so it has to be given in his daily diet if he is to have plenty.

About three years ago I raised in the neighborhood of twenty puppies that never nursed their mother and I never lost a puppy out of that bunch. I kept them for two reasons. The owners didn't want them and I didn't want to put them to sleep. After a while I thought it was a good way to keep them from breaking distemper.

We follow that method with all of our baby puppies and have very little trouble. I would advise it.

I had a litter of ten bird dog puppies. Before they had their eyes open they were eating about a pound of raw beef and about a quart of raw milk besides what the mother gave them every day. In two weeks they were eating two pounds of raw meat. Before their eyes were really open, they would crawl to a pan of meat we would set in the room. They gained in weight and in hard bone.

I would like to say something about potatoes. We always call Monday "wash day." Sunday people have their large Sunday dinners in the middle of the day or afternoon and about sundown or bed-time they call up and say, "Doctor, come quick! My dog has gone mad!" When you ask them how much potatoes they fed the dog that day they will tell you they gave him "what was left from dinner."

We give them a stomach wash, and I think I am safe in saying that ninety per cent of them will have plenty of potatoes, mashed, whole and otherwise, and I also think I am safe in saying that less than ten per cent of those that have spasms have meat. We are of the opposite opinion of this. We have less digestive troubles. In cases of non-parasitic skin diseases we still stick to meat. We have put a few on vegetables but couldn't arrest the condition.

I would suggest that you try the Carnation milk and cod-liver oil, leaving out lime water. It was only a few days ago that I read an article by some research man who said calcium was not assimilated without vitamin D.

DR. SCHLOTTHAUER: Don't think that I am a vegetarian and that I recommend vegetables entirely. I recommend a mixed diet for economical and health reasons. Some people have fed dogs nothing but meat and had very bad results. I recommend meat in all diets for all dogs, whether Pekingese or bird dogs. I would say one-fourth meat for all of them is a minimum content. I am getting by with that. Some of you will not get by with that amount. Some of you, on the other hand, will get by with things that I can't. Some of you will take into consideration certain conditions in dogs that we do not find.

In regard to the statement that potatoes caused convulsions—that is purely the result of overloading of the stomach. That is too soon for any toxic effect from potatoes to develop. People overload their stomachs, too. The dog has been going all day and has to wait longer than the people to eat and naturally he is hungry. If a lot of food is placed before him, he will eat it, and puppies, especially, will have regular fits from eating too much.

DR. T. H. FERGUSON: I would like to ask Dr. Schlotthauer the amount of milk and glucose that he feeds a sick dog by tube in proportion to the size of the stomach, whether a quarter full, or half full, or just a small amount.

DR. SCHLOTTHAUER: It is better to give small amounts than large. For a police dog a pint would be ample. He will have a better chance of retaining it than if you give him a large amount and much of it will go through undigested.

DR. O. B. MORGAN: I hear some of the gentlemen mentioning a milk diet. I know a woman who feeds milk to Pomeranians, but in place of diluting the milk with water, she adds cream to it, with very good results. I wonder if any of you have done that.

DR. SCHLOTTHAUER: They can do it if they want to; it costs a little more.

I think the discussion brings out my early contention, that a dog is adaptable. You can feed almost anything and get away with it.

DR. J. V. LACROIX: There is always diversity of opinion. Regarding the use of a greater amount of fat in cow's milk than exists normally, I suggest you add an egg yolk, using it either raw or slightly cooked, beating it with the milk and setting that away in the ice-box, giving the proportion recommended by Dr. Blamey, of New York City, one egg yolk to a teacupful of milk. The egg yolk gives vitamins A and B, also minerals and fat. This, of course, is given at room temperature.

Regarding the weaning puppy, or one that does not have its mother to suckle, and the use of sugar, I have had occasion to make observations over a period of eight years, and I think that cane sugar is superior to corn sugar. Dr. Young spoke of milk containing sugar. I think it is evaporated milk that contains the sugar, although I am not clear on that. At any rate, I believe, the cane sugar is superior to the corn sugar, particularly in distemper-affected puppies. Corn sugar is immediately available as a fuel and has an advantage, perhaps, in forestalling dehydration.

DR. SCHLOTTHAUER: I don't know whether cane sugar has any greater value than corn sugar or not. I know the leading pediatrists throughout the country recommended putting white Karo syrup in baby's milk, and I think we all agree babies are about as delicate as any animal.

DR. F. F. PARKER: Dr. Schlotthauer, your paper has brought out splendid discussion, and it would appear that two things, at least, had been proved. One is that the dog should have all or the majority of its food in large pieces. We all know he does not chew his food and that is one of the secrets of his digestion.

DR. HAMLET MOORE: There is one thing that has not been brought out that I would like to have discussed, that is, what about water in feeding? When and how should water be given the dog? No one has mentioned that.

DR. SCHLOTTHAUER: We have water before the dogs at all times. We let them drink as much as they will. A dog has a peculiar digestive tract, with a large stomach and short intestinal tract. If you deprive him of water for a long period of time so that he gets very thirsty and then give him food and water, he could easily upset the digestion of food in the stomach by overloading with water. It would dilute the acid to such an extent it would be ineffective.

AN EPIZOOLOGICAL STUDY OF SHIPPING FEVER IN KANSAS*

By HERMAN FARLEY, *Manhattan, Kansas*

Kansas Agricultural Experiment Station

A study of shipping fever in Kansas during the past two years has resulted in a few important preliminary observations on that important problem.

An experimental project on shipping fever in cattle was started in 1929 and investigations of between 100 and 200 outbreaks of the disease, on as many farms, located in most parts of the State, have been made. The conditions as to housing, feeding and watering and the history of the affected herd were obtained. A careful examination of all sick animals was made and autopsies performed whenever possible.

From a study of the conditions of housing and care given animals on farms where the disease occurred, it was soon evident that the number of sick animals was always higher when inadequate shelter was provided for recently shipped-in animals. The number of sick animals and the proportion of deaths was highest when inadequate shelter was accompanied by carelessness in the feeding and watering of the animals. In four cases where inadequate shelter and poor feeding conditions prevailed, native cattle were confined in close quarters with the sick shipped-in animals; some of these native cattle sickened with clinical cases of shipping fever. In other cases where native cattle came in contact with cases of shipping fever, no symptoms of the disease developed in the native stock.

The incidence of shipping fever was greatest during wet, cold weather, especially during changeable spring and fall weather. Table I shows that losses were highest during October and November and were again high in March. The severe weather conditions of the fall coincide with the heaviest shipping of feeder cattle.

The length of the rail transportation during the wet, cold months had apparently some correlation with the severity of the disease as it manifested itself on the farm. Especially heavy losses occurred among cattle shipped from southern Texas and from Canada during October and March. In at least one of these cases

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TABLE I—*Losses from shipping fever in unvaccinated and vaccinated cattle*

MONTH	NOT VACCINATED				VACCINATED				WEATHER
	ANIMALS	HERDS	DEAD		ANIMALS	HERDS	DEAD		
			No.	%			No.	%	
1930									
October	404	3	9	2.2	718	8	19	2.6	Rain
November	963	11	11	1.1	1249	18	38	3.4	Snow—rain
December	1357	8	5	0.3	1052	17	40	3.7	Snow—rain
January	596	7	6	1.0	1232	12	39	3.1	Cold—fair
February	149	4	3	2.0	684	7	19	2.8	Fair—cool
March	217	3	2	0.92	292	5	6	2.5	Changeable
April	212	1	1	0.47					Mostly fair—warm
July					100	1	6	6.0	Hot—dry
August	70	1	2	2.85	133	2	6	4.5	Hot—dry
1931									
January	126	2	1	0.70					Clear—cold
February	25	1	2	8.0					Mostly fair—warm
March					201	1	30	14.8	Changeable
Total	4119	41	42	1.02	5661	71	203	3.58	

TABLE II—*Distribution of shipping fever in animals treated and untreated in the stockyards*

	Not Vaccinated at Yards									Vaccinated at Yards								
	Not Treated at Farm		Number of Herds	Treated at Farm						Not Treated at Farm		Number of Herds	Treated at Farm					
				Bacterin		Aggressin		Serum					Bacterin		Aggressin		Serum	
	No.	Dead		No.	Dead	No.	Dead	No.	Dead	No.	Dead		No.	Dead	No.	Dead	No.	Dead
1930																		
Oct.	401	8	3					3	1	649	15	8	69	4				
Nov.	842	17	10	180	7			121	0	1021	28	18	48	3				
Dec.	899	5	7	540	29	93	6			325	14	17			90	9		
Jan.	591	5	7	260	3					939	16	12						
Feb.	149	3	4	44	0					640	33	7						
March	217	2	3	182	5					110	1	4						
April	212	1	1															
July			1	100	6													
Aug.	70	2								70	1	2	63	5				
1931																		
Jan.	124	1	2					2	0									
Feb.	25	2	1															
March												1	201	30				
Total	3530	46	39	1306	50	93	6	126	1	3754	108	69	381	42	90	9		
Per-centage		1.30			3.82		6.46		0.71		2.87			11.2		10		

delays in receipt and movement of the cattle occurred. Direct shipments of cattle to the feeder from the ranch on which they were raised and without being handled at local or public stockyards had fewer clinical cases; but even under these conditions at least one such shipment suffered a severe outbreak of shipping fever.

A careful history of the condition of the shipped-in animals was obtained and it was ascertained whether any or all the animals had been vaccinated before shipment, or at the stockyards, and whether any biological products had been used since arrival. Table I shows a comparison of the losses incurred from shipping fever among animals that had been vaccinated at the yards or on the farm, and among untreated animals. The nature of the product used at the stockyards could not be ascertained except in a few cases, so all vaccinations, whether with bacterin, mixed vaccines or aggressin, are listed under one head. In table II the various products used on the farms are listed separately.

Losses Greater in Vaccinated Animals

Table I shows that losses among vaccinated cattle were about three times as high as among untreated animals. Among 5661 vaccinated animals there was a loss of 3.58 per cent and among 4119 untreated animals the loss was 1.02 per cent. The losses were higher in the vaccinated group during every month in which outbreaks occurred and in only one month, February, when the weather was fair and cool, did the losses in untreated animals approach the losses among the vaccinated group.

Table II indicates the incidence of shipping fever among herds vaccinated at the stockyards, or at the farm, and also lists the vaccine used at the farm. The product used at the stockyards could not be determined with any certainty.

From a study of this table it is seen that among a rather small number of animals vaccinated at the yards and treated a second time at the farm, the losses were very high. In 381 animals vaccinated at the yards and given bacterin at the farm the losses were 11.2 per cent, and among 90 head vaccinated at the yards and given aggressin at the farm the loss was 10 per cent. Animals treated with aggressin at the farm showed a loss of 6.46 per cent in 93 head. Of 1306 cattle given bacterin at the farm the loss was 3.82 per cent. The losses among the cattle vaccinated at the yards and untreated at the farm were 2.87 per cent in 3754

head and the untreated animals showed a loss of 1.30 per cent in 3530 head studied.

It was seen that vaccination of animals at the farm after they have been subjected to the trials and exposures of a rail journey was followed by a slightly greater proportion of loss than where vaccination was performed at the yards. Double vaccination, at the yards and again at destination, proved very costly in the comparatively few cases in which this was practiced.

The use of anti-hemorrhagic septicemia serum apparently gave very good results. In two cases the serum was given to very sick animals. In one herd three animals were treated with the loss of one; in a second, two sick animals were treated with no subsequent deaths and both recovered. One herd of 121 head, which showed evidences of shipping fever in 20 per cent of the animals, was treated with serum and none of this herd died.

Size of Calves Not a Factor

Table III classifies the cattle according to weight. In this table, as in the two previous tables, the losses among the vaccinated group are higher than the losses with the untreated animals. The losses are higher for the vaccinated groups in each weight classification. For example, considering animals between 200 and 300 pounds, there were no deaths among 184 untreated animals and 5.8 per cent in 293 vaccinated cattle. In the case of 500- to 600-pound animals, 496 untreated cattle showed a loss of 0.6 per cent, and 749 vaccinated heavy calves of this weight, a loss of 5.6 per cent. The number of deaths in the increasing weight classifications shows only a very slight decrease and it would seem that the size of the calves has only a very slight correlation with the incidence of shipping fever.

Table III—*Losses from shipping fever in cattle of different weights*

Weight (lbs.)	Not Vaccinated			Vaccinated		
	No.	Dead		No.	Dead	
		No.	Per Cent		No.	Per Cent
200-300	184	0	0	293	17	5.8
300-400	919	16	1.7	2034	106	5.2
400-500	441	7	1.58	749	26	3.5
500-600	496	4	0.8	749	42	5.6
600-700	1113	7	0.6	176	10	5.7
700-800	192	4	2.0	76	3	3.9
Over 800	255	4	1.5			
Total	3600	42	1.16	4077	204	5.0

In a report of the Committee on Miscellaneous Diseases of the U. S. Live Stock Sanitary Association,[1] the Committee reports an experiment on the vaccination of a number of cattle in the stockyards. A series of 151,457 cattle were vaccinated at the yards and losses of 662 (1.14 per cent) were reported in 57,946 head from which reports were obtained. This report states that losses among untreated animals were the same. Among 708 animals given aggressin, the loss was 40 (5.64 per cent) and of 700 untreated, 17 died (2.42 per cent). This Committee concludes that there is no protection developed during the incubative stages of the disease but that immunity may be conferred if the products are given 10 days before shipment. Buckley and Gochenour[2] found that vaccinated animals are more susceptible to infection with *Pasteurella boviseptica* than unvaccinated animals. Resistance to the disease does not begin until from the sixth to the ninth day.

Record of Three Lots of Yearlings

The record of three lots of short yearling cattle is of sufficient interest to report in some detail. Three neighbors purchased 80 head of cattle each from the Wichita stockyards during October, when the weather was good. These cattle were sorted out from various lost in the yards and were then shipped to destination together. The first lot was driven three fourths of a mile on arrival, and placed with nine native cattle in fairly good quarters. Three days later some ten head sickened with shipping fever. These animals were treated with intestinal antiseptics and stimulants; all the herd was given an injection of hemorrhagic septicemia bacterin. Two of the sick animals died within the next five days, when the herd was again bacterinized and the sick animals treated. This second treatment was followed by a loss of three more animals.

The second lot of 80 head was driven one and one-half miles. Three days later, this lot also showed a number of cases of shipping fever. Twenty head were treated with intestinal antiseptics and stimulants and were given hemorrhagic septicemia bacterin. Two of the treated animals died during the next ten days.

The third lot of 80 head was driven five and one-half miles. In this lot, 40 animals sickened between the third and tenth days. No treatment of any kind was used in this herd and all animals recovered.

Summary

1. Losses among vaccinated cattle involved in this study were higher than among unvaccinated animals.

2. Shipping fever was more severe in cold, wet weather, and the increase of loss during bad weather was greater among vaccinated than with untreated animals.

3. The weight of the animals and losses from shipping fever were not correlated. Heavy animals seemed to be as susceptible as 200-pound calves. Losses were heavier in all weights of vaccinated animals than in the unvaccinated animals.

4. Vaccination of animals at the farm after shipment was followed by a slightly greater loss than when vaccination was practiced at the yards.

5. Vaccination of cattle at the farm after they had been vaccinated at the yards resulted in very heavy losses (10 and 11 per cent).

6. Treatment of a small number of cattle with anti-hemorrhagic septicemia serum resulted in a reduction of losses to a very small figure.

References

[1]Miller, A. W., et. al: Report of Committee on Miscellaneous Transmissible Diseases of the U. S. Live Stock Sanitary Asso. Jour. A. V. M. A., lxx (1927), n. s. 23 (6), pp. 952-955.

[1]Buckley, J. S. and Gochenour, W. S.: Immunization against hemorrhagic septicemia. Jour. A. V. M. A., lxv (1924), n. s. 19 (3), pp. 308-311.

Discussion

Dr. A. E. Cameron: Have you any information on the time that the cattle were vaccinated on the farm before shipment. I think you mentioned that some were vaccinated on the farms.

Dr. Farley: From our field observations we really did not have any definite records on animals that were shipped from their point of origin. This number of cattle included principally those animals shipped from the stockyards and we couldn't trace the animals from their point of origin.

Dr. Cameron: Generally speaking, vaccination is practiced while the animals are debilitated or exposed to the disease. They are much more likely to have an attack of the disease when aggressins or live cultures, particularly, are used and seemingly the best results will be obtained when vaccination against hemorrhagic septicemia is practiced a week or ten days before exposure.

Dr. F. H. Brown: It has always been our conception that if these cattle could be vaccinated ten days or two weeks before they enter the channels of trade, our losses would be reduced materially. It has also been our observation that the use of anti-hemorrhagic septicemia serum on infected animals was worth while, especially if we were dealing with the dairy or breeding type of cattle.

Dr. W. T. Spencer: I think the experience related in Kansas relative to the more severe infection in cattle coming through the stockyards is a little different from that of the United States Bureau of Animal Industry. I think that Dr. Miller, in his statement in Chicago, a year or two ago, said in effect that cattle handled through the public stockyards were subject to a smaller loss than the ones that had been handled through other channels.

I think those of us who are familiar with the manner in which cattle are handled at the public stockyards and who know the conditions of the pens and everything into which the cattle are loaded can appreciate that fact. There are fewer chances for infection in the handling of feeder cattle through the public stockyards than through the average feed-yard. I know that is true in

our section of the country, because we have feed-yards in Nebraska where a great many feeder cattle are handled, and there is practically no supervision over those yards, and I think that the public stockyards, all of them, are very careful in the handling and care of the cattle that are going to pass through those yards for feeder purposes.

The yards are cleaned carefully and disinfected regularly under federal supervision. I think a man is taking less chance of infection by buying his cattle through the public stockyards than through the average feed-lot through which they pass in direct shipments.

I want to bring that point out, because I think it is important, and every effort is being made at the public markets to safeguard the handling of feeder cattle. I understand the Bureau of Animal Industry is contemplating, in fact already has under way, an extensive program of investigation to determine these points raised here as to the time of vaccination and as to whether any of the vaccines are of value or not. The public stockyards are going to offer all the assistance in the way of coöperation they can to the Bureau of Animal Industry in carrying on that experiment.

DR. J. W. CONNAWAY: Dr. Spencer may also recall that Dr. Miller, in giving his report, gave no encouragement to vaccination in the stockyards before shipping the cattle out.

DR. A. H. FRANCIS: That is true, and I can say the men at the yards are giving no encouragement. The experience of the commission men—and I have had close contact with them—is that they are advising their customers not to have them vaccinated.

DR. J. P. SCOTT: Considering the four cases reported as direct shipments, I think perhaps there is a little misunderstanding. These four cases were not handled through any feed-yards, but were bought by the buyer from the original owner.

DR. CAMERON: I think most of us will agree with Dr. Spencer that the cattle are quite favorably handled in public stockyards, but unfortunately the trouble is that the disease usually develops before the cattle reach the stockyards. It is probably the exposure en route. Some of them travel long distances, and the fact is that numbers of organisms may be found in normal cattle and appear to develop for reasons which are not understood at all. Consequently the only protection likely to be effective is vaccination in time for the vaccination to become effective before the cattle are loaded on the trains.

DR. CAMPBELL: There is one thing that occurs to me in regard to this matter. In travelling through the country, which we do, testing cattle for tuberculosis, we find cattle with shipping fever, so-called, that have never been shipped at all. They have it on the farm where they are raised. Of course, if you load those cattle on the train and ship them to market, you will have this condition there and that probably will be intensified.

We frequently find it on a farm where no cattle have been shipped at all, and there is sometimes quite a bit of loss.

There is a question I would like to ask. Can you immunize against a disease that does not itself confer immunity? We know when a hog recovers from hog cholera, that hog is immune. It will never have hog cholera again, and on that basis we vaccinate and do immunize hogs. We have not determined, I think, whether shipping fever recurs the second time or if one attack does confer immunity. If it does, all right. Vaccinate them. If it does not confer or produce immunity to a disease that does not immunize the animal itself, it will do no good.

DR. C. H. HAYS: In regard to Dr. Campbell's statement, the limited observation we have had indicates there are certain influences that have a bearing in the cases that occur where, apparently, at the time there was no reason for it, such as have been indicated here—long-distance shipments, young cattle on the range, and so on. It is generally found that there has been some fatiguing influence, such as driving. In the feed-lot and on farms, dietetic errors have been found and observed.

DR. CAMERON: On immunity in hemorrhagic septicemia, I believe I am safe in saying that experiments have established the fact that aggressin will be effective. It would be practically impossible to prove immunity against the natural disease. Losses vary from a very small number up to about fifty per cent.

PRELIMINARY BACTERIOLOGICAL REPORT ON SHIPPING FEVER*

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Material for this investigation was taken from autopsies made during the field study of 125 herds of cattle affected with shipping fever. In twenty-six of these herds autopsies on one or more cattle were made and material was brought back to the laboratory packed on ice.

The characteristic lesions of croupous pneumonia, petechiae on serous membranes, hemorrhagic areas in the subcutis, and gastro-enteritis were found in all the cases examined. Portions of lung, liver, kidney, spleen, muscle or subcutaneous connective tissue, and heart-blood and peritoneal or pleural exudates were shipped or brought to the laboratory as soon as the autopsy was completed.

Isolation of organisms from this material was made on salts agar,[1] consisting of:

Agar	15 to 20 grams
Ammonium phosphate	0.5 grams
Potassium bicarbonate	0.5 grams
Potassium citrate	2.0 grams
Glucose	0.5 grams
Glycerol	5.0 grams
Sodium chlorid	2.7 grams
Beef heart decoction	1000.0 cc

To maintain or increase virulence, 2.5 grams of ferric ammonium citrate was added or substituted for the sodium chlorid. This agar medium does not require filtration and its reaction is about pH7. It is well to check this and adjust it to pH 7.2. If hemophilic organisms were present or suspected, horse or rabbit blood was added to the sterilized medium.

Rabbit and guinea-pig inoculations also were made from the heart-blood or exudates.

Three general groups of organisms were found in the examination of material from these cases. The most constant organism found was *Pasteurella boviseptica*, which was isolated from 21 (80.77 per cent) of the 26 cases examined. Pasteurella cul-

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tures were recovered also from routine autopsies in four cases: First, from a case of necrotic laryngitis, where the organism was found in the necrotic material, which also contained *Actinomyces necrophorus*. Second, from the lungs of a calf, which died of bloat. Third, from the lungs of a sheep, dead of overfeeding disease. Fourth, from a steer, which had been rapidly brought on to full feed containing large amounts of cottonseed cake.

The second most common organisms were of the colon-aerogenes type. These were isolated in 10 (38 per cent) of the cases. Colon-type cultures were found in all material which took more than 15 to 24 hours to reach the laboratory. In most cases where the material reached the laboratory promptly, the only organism isolated was *P. boviseptica*.

The third class of organisms found were gram-negative rods which did not ferment any of 22 carbohydrates examined. A number of these organisms were compared with *Alcaligenes bronchisepticus* and found to correspond culturally and antigenically with this organism. Ten of these cultures were found and six of them belonged to the *A. bronchisepticus* species.

Organisms resembling diphtheroids were found in three cases and organisms producing acid, but no gas in all carbohydrates tested, were isolated from three other autopsies.

The identification of *P. boviseptica* was made by comparing the morphological, fermentative and pathogenic characters of the organisms isolated with the characters of twelve cultures of *P. boviseptica* obtained from three commercial laboratories, three experiment stations, the Bureau of Animal Industry and from India.

The fermentative characteristics of the Pasteurella cultures, the colon-aerogenes types and other unknown organisms were determined on the "salts sugar free" medium[1] consisting of:

Distilled water	1000.0 cc
Peptone	20.0 grams
Ammonium dihydrogen phosphate	0.5 grams
Potassium bicarbonate	0.6 grams
Ferric ammonium citrate	2.5 grams
Disodium hydrogen phosphate	1.7 grams
Agar	1.0 grams

To this 1 cc of a ten per cent solution of the carbohydrates was added after sterilization.

It was found that four characteristics were sufficient to make a classification of Pasteurella cultures.

TABLE I—*Bacterial flora found in cases of shipping fever*

PASTEURELLA BOVISEPTICA	CASES	COLON AEROGENES	CASES	ALCALIGENES BRONCHISEPTICUS	CASES
Alone	5	Alone	0	Alone	0
Associated with:		Associated with:		Associated with:	
E. coli	6	*P. boviseptica*	6	*P. boviseptica*	4
A. bronchisepticus	4	*A. bronchisepticus*	3	*E. coli*	3
E. coli and *A. bronchisepticus*	1	*P. boviseptica* and *A. bronchisepticus*	1	*P. boviseptica* and *E. coli*	2
Acid-former	1	*P. boviseptica* and diphtheroid	1	*P. boviseptica* and acid-former	1
E. coli and diphtheroid	1	*P. boviseptica* and acid-former	1	Diphtheroid, *E. coli* and *P. boviseptica*	1
E. coli and acid-former	2			*E. coli*, acid-former and *P. boviseptica*	1
A. bronchisepticus and acid-former	1				
Totals	21		12		12

1. Staining reactions of smears from the tissues showed bipolar organisms when stained with methylene blue.

2. The colonies developing on agar were small, translucent and not over 2 to 4 mm. in diameter.

3. The organisms found in smears from agar or broth cultures were short, slender, gram-negative rods occurring singly.

4. The cultures were pathogenic for guinea pigs or rabbits when first isolated, or after repeated transfer on ferric-salts agar.

Table I summarizes the bacterial flora found in the 26 cases of shipping fever. Table I shows that *P. boviseptica* was the most common organism found and that it was the only organism to be isolated alone from material obtained in cases of shipping fever.

P. boviseptica was isolated from the lung tissue alone in 7 of the 21 cases; from both heart and lung in 5 autopsies; from the heart alone in 4 and from animal inoculation in 5 cases. In the 5 cases where animal inoculation was necessary for the isolation of Pasteurella cultures, colon-type organisms were found on direct isolation.

Examination of Normal Lungs

Portions of 83 normal lungs were obtained from a packing-house in Kansas City and were shipped to Manhattan packed in ice. These lungs were carefully examined by cultural methods and the bacterial flora determined. None of these lungs yielded any cultures of *P. boviseptica*; colon organisms were found in 8 cases and *A. bronchisepticus* or related organisms in 8 cases. Diphtheroid-like organisms also were isolated in 8 cases. The most common organisms were staphylococci and streptococci of various species.

From a study of the bacterial flora found in cases of shipping fever and from normal calf lungs, it would seem that *P. boviseptica* was the most important bacteriological factor in shipping fever.

Spray,[2] in examining 100 normal lungs of swine, found *P. suiseptica* in 4 of these lungs. In examining 314 pneumonic lungs, he isolated *P. suiseptica* in 5 per cent of the lungs examined. *B. alcaligenes* was found in 3 normal lungs and in 3 per cent of the pneumonic lungs. Spray also found an inulin-fermenting streptococcus in 12 per cent of normal lungs and 35 per cent of pneumonic lungs. Schalk[3] found *P. suiseptica* in 3

normal lungs and 3 pneumonic lungs, out of 12 hog-cholera pigs examined. Jorgensen[4] studied the bacterial flora of the nasal passage of 250 normal cattle. He isolated *P. boviseptica* from 37 cases, and concludes that *P. boviseptica* is a normal inhabitant of the upper respiratory tract of cattle. After feeding experiments on cattle, one cow was chilled and driven for one hour and then sprayed on the nose with cultures of *P. boviseptica;* this treatment resulted in a congestion of the lungs and diarrhea. A second cow was chilled, driven seven miles and then fed *P. boviseptica* cultures. This animal died of clinical shipping fever. From these experiments Jorgensen concluded that *P. boviseptica* could become pathogenic when animals were exposed to the elements.

The isolation of *P. boviseptica* from four general autopsy cases which had no visible lung lesions suggests that while *P. bovi-*

TABLE II—*Bacterial flora of 83 normal calf lungs*

SPECIES	TIMES ISOLATED	LUNGS CONTAINING ORGANISM (%)
Staphylococci	36	43
Streptococci	33	39
Gram-positive rods, free-growing	26	31.3
Diphtheroids	8	9.6
Colon-aerogenes group	8	9.6
A. bronchisepticus	8	9.6
Spore-forming organisms	7	9
Cocco-bacillus	4	4.9
Pasteurella	0	0

septica may be the primary bacteriological factor of shipping fever, the causative factor in the production of this disease should be looked for elsewhere.

IMMUNIZATION STUDIES

Rabbits were injected with from 5 to 10 injections of saline suspensions of agar cultures of 10 strains of *P. boviseptica,* at intervals. After the last injection the sera from these rabbits were used to determine the agglutination reactions of 32 cultures of *P. boviseptica.* Dilutions from 1:40 to 1:1600 were used. The tubes were incubated for 12 hours and then placed in the ice-box for 12 hours. Occasional tubes required a second incubation and refrigeration before a reading could be obtained.

It was found that two cultures were agglutinated in high dilutions by a majority of the ten sera used. There was no

TABLE III—*A comparison of the agglutination titres obtained in rabbits by injections of P. boviseptica and their survival following an injection of P. boviseptica culture*

RABBIT	CULTURE	IMMUNIZATION			AGGLUTINATION	DATE	TEST INJECTION			RESULT
		INJECTION		LAST INJECTION DATE	TITRE FOR CULTURE 17		CULTURE	DOSE (CC)	DATE	
		NO.	DOSE (CC)							
724	18	8			1:320					D. 5 days
10	17	5			1:1600					D. 5 days
897	17	5			1:1600					D. 5 days
898	17	1	10	4–4	1:80	5–1	17	0.12	5–1	Lived
829	1	1			1:160					D. 4 days
895	1	1			0					D. 3 days
834	17	1			1:160					Lived
839										D. 2 days
1	1 B	1			1:40					D. 2 days
2	1 B	1			1:80					D. 4 days
3	17 B	1			0					D. 7 days
4	17 B	1			1:320					Lived
5	1 KB	1	5	5–10	1:80	5–22	17	0.05	5–22	D. 2 days
6	1 KA	1			1:160					Lived
7	17 KB	1			1:80					Lived
8	17 KA	1			0					Lived
9	17 KA KB	1			1:40					D. 2 days
10										D. 18 hours

B = broth culture antigen.
A = saline emulsion of agar culture.
KB = broth culture heated to 100° C. for 40 minutes.
KA = saline emulsion of agar culture heated to 100°C. for 40 minutes.
KA KB = Agar emulsion washed off with broth culture and boiled for 40 minutes.

clear-cut differentiation into grcups as suggested by Jones.[5] Three subdivisions of the 32 organisms could be recognized if the criterion of separation used was that the strains should agglutinate in the same dilution as the homologous culture.

The correlation of the degree of agglutination obtained by repeated injections of agar cultures, of single injections and of the protection developed against injections of *P. boviseptica* were studied. Table III shows the results obtained from the injection of rabbits of known agglutination reaction against the culture used for the agglutination and test injection.

The agglutination reaction of the sera was determined using dilutions from 1:40 to 1:1600. Table III shows that there was no direct or constant relationship between the degree of agglutination developed in the rabbit and its survival following a test dose of virulent culture. There was some immunization shown if the survival time of the immunized rabbits is taken into consideration. Rabbits 10 and 897, having agglutination titres of 1:1600, died in five days, whereas rabbit 898, having a titre of 1:80, survived. Rabbit 2, having a titre of 1:80, died in 4 days and rabbit 3, having a titre of 0, lived for 7 days. Rabbit 4, having a titre of 1:320, survived.

A series of rabbits and guinea pigs were inoculated with various broth and agar suspension bacterins made from cultures of *P. boviseptica* and with commerical products. None of the products tested showed any satisfactory grade of immunizing power. Out of 123 guinea pigs injected with 2 to 5 cc of various *P. boviseptica* antigens, 87 (70.7 per cent) died during the five days following the test injection. Seventy-three rabbits were immunized and tested in ten days to two weeks with virulent cultures of *P. boviseptica;* 65 (89 per cent) of these animals died.

The degree of virulence of the test culture had a marked effect on the percentage of survival. If the test dose of culture killed the control guinea pig or rabbit in less than 15 hours, the percentage of surviving test animals was less than 5 per cent. If the test dose took more than 24 hours to kill the control guinea pig or rabbit, the survival percentage was from 5 to 15 per cent for rabbits and 10 to 20 per cent for guinea pigs, thus showing some immunization.

Reports of the Japanese and German workers on boiled antigens, the formulation of the Impedin theory by Torikata in 1917 and reviews in more recent papers[6] suggested that boiled broth cultures mixed with agar suspensions might prove of value.

A series of 32 rabbits were inoculated with 48-hour broth and agar cultures killed by heating to 58 degrees and also with boiled or Kocto antigens from broth and agar cultures of a virulent culture (17) and an avirulent culture (18) which had immunized rabbits previously.

TABLE IV—*Immunization of rabbits with Pasteurella antigens*

Rabbit	Weight (grams)	Injection			Test Injection			Result
		Material	Dose (cc)	Date	Culture No.	Dose (cc)	Date	
11	1500	17 KB F	5					D. 3 days
12	1500	17 KB F	10					D. 4 days
13	1600	18 KB F	5					D. 4 days
14	1500	18 KB F	10					D. 1 day
15	1700	17 B F	5					D. 5 days
16	1500	17 B F	10					D. 2 days
17	1500	18 B F	5					D. 1 day
18	1600	18 B F	10					D. 4 days
19	1400	17 KB	5					Lived
20	1500	17 KB	10					D. 2 days
21	1500	18 KB	5					D. 1 day
22	1500	18 KB	10					D. 2 days
23	1500	17 B	5					D. 1 day
24	1500	17 B	10					D. 5 days
25	1500	18 B	5					D. 4 days
26	1500	18 B	10					D. 1 day
27	1400	17 KA	3	6–16	17	0.025	6–26	D. 1 day
28	1500	17 KA	3					D. 2 days
29	1500	18 KA	3					Lived
30	1500	18 KA	3					D. 2 days
31	1500	17 A	3					D. 4 days
32	1500	17 A	3					D. 3 days
33	1500	18 A	3					D. 10 days
34	1500	18 A	3					D. 3 days
35	1500	17 KB A	3					D. 2 days
36	1500	17 KB A	3					D. 2 days
37	1500	18 KB A	3					D. 2 days
38	1500	18 KB A	3					D. 2 days
39	1500	17 B A	3					D. 7 days
40	1500	17 B A	3					D. 2 days
41	1500	18 B A	3					D. 3 days
42	1500	18 B A	3					D. 3 days
43	1500				17	0.025	6–25	D.15hours
44	1500				18	0.75	6–10	Lived
45	1800				17	0.025	6–10	D. 1 day

KB = Kocto broth.
F = salts broth containing ferric ammonium citrate.
KA = Kocto agar.
B = broth culture.
A = agar culture.

The antigens used in table IV included boiled broth cultures or Kocto broth antigens (KB)[6] from cultures grown in salts broth and salts broth containing ferric ammonium citrate to increase the virulence of the cultures. Two cultures were used, a virulent

culture (17) and culture 18 which had proved antigenic. At the time the experiment was started, culture 18 did not kill rabbits in doses of 0.75 of a whole ferric broth culture. From the results obtained in this test it would seem that when a strongly virulent culture is used as a test of immunity there is very little immunity produced and that none of the different products tested proved of any value. The two rabbits that survived were inoculated with boiled broth culture 18 and boiled saline suspension of culture 18. This suggests that this avirulent culture is more antigenic than the virulent culture 17.

Tanaka[7] and Morch[8] show that Pasteurella organisms are only very slightly antigenic either for agglutination and complement-fixing substances or for the immunization of animals. Morch suggests that sanitary measures are the only measures

TABLE V—*Susceptibility of rabbits and guinea pigs to Pasteurella boviseptica (culture 17)*

RABBITS (1500 GM.)			GUINEA PIGS (350 TO 450 GM.)		
DOSE	DATE	RESULT	DOSE	DATE	RESULT
0.1		D. 3 days	0.3		Lived
0.05		D. 4 days	0.3		D. 1 day
0.025		D. 2 days	0.2		D. 2 days
0.025		Lived	0.2		Lived
0.012		D. 6 days	0.1		Lived
0.012	7–2	Lived	0.1	7–11	Lived
0.006		D. 2 days	0.05		D. 2 days
0.006		Lived	0.05		Lived
0.003		D. 5 days	0.025		Lived
0.003		D. 2 days	0.025		D. 1 day
			0.012		D. 3 days
			0.012		Lived

that can be expected to reduce the losses incident to shipping. Roderick[9] finds that there are two types of Pasteurella organisms, the bovine-swine group and the ovine-avian-rabbit type, which is the more pathogenic.

A series of 10 rabbits and 12 guinea pigs were injected with graduated doses of culture 17 and the results are given in table V.

From a study of table V it is seen that rabbits and guinea pigs vary quite markedly in their susceptibility to injections of Pasteurella cultures. Doses three to eight times as large as the smallest dose sufficient to kill some rabbits was not enough to kill other rabbits of the same age and weight. The guinea pigs required a higher dosage, but the variability is apparent in these animals also.

Calf Infection Experiments

Seven calves were obtained and inoculated with 48-hour agar culture emulsions corresponding to tube 4 of the McFarland nephelometer.

Calf 1: Injected subcutaneously with 20 cc of *P. boviseptica*, strain 8. This calf showed a rapid increase in temperature, bloating, and was dead in 18 hours. Autopsy revealed extensive subcutaneous hemorrhages, petechiae on serous surfaces, lungs injected and edematous. Rumen, abomasum and intestines, extensive areas of inflammation and extremely friable throughout.

Calf 2: Injected intravenously with 22 cc of *P. boviseptica*, strain 28. Dead in 15 hours. Symptoms and lesions identical with calf 1, with the exception that petechiation of serous membranes was absent.

Calf 3: Injected intravenously with 20 cc of *Escherichia coli*. Rapid rise in temperature and bloat. Dead in six hours. Autopsy revealed a few subcutaneous hemorrhages, petechiae on serous surfaces, extensive gastro-enteritis and injection of the lungs.

Calf 4: Injected subcutaneously with 12 cc of *P. boviseptica*, strain 28. Temperature increased from 101.4 to 105.2° F. in five hours; some stiffness and inappetence. Animal almost normal within 24 hours. Second injection of 20 cc of emulsion of *P. boviseptica* given at this time. Slight rise in temperature and complete recovery within three days.

Calf 5: Animal fed ordinary hay and grain ration for three weeks and kept in the Veterinary Hospital. Injected with 15 cc of *P. boviseptica* culture. Temperature increased from 101.3 to 105.8° F. in five hours, stiffness, inappetence and death in 18 hours. Autopsy revealed subcutaneous hemorrhages, petechiae of serous surfaces, injection and edema of lungs. Extensive gastro-enteritis. Intestines very friable. Rumen contents dryish.

Calves 6 and 7: Grain and hay withheld for 24 and 48 hours before injection of 15 cc of *P. boviseptica* emulsion. Increase of temperature: calf 6, 101.6 to 105.2; calf 7, 101.6 to 102.2° F. Temperatures normal in 24 hours. Recovery rapid.

Ten days after first injection, calf 6 was injected with 20 cc of a culture of *E. coli*. Rise in temperature from 101.2 to 102° F. in four hours. Recovery to normal condition in 24 hours.

These experiments indicate that calves can be killed by intravenous injections of normally pathogenic and non-pathogenic organisms, and that *P. boviseptica* can cause death in calves on moderate feed, but that calves fed small amounts of hay and grain and plenty of water resist injections of *P. boviseptica* given subcutaneously. *E. coli* did not kill a calf previously injected with *P. boviseptica*.

From the bacteriological examinations of material from cases of shipping fever and from normal lungs it would seem that *P. boviseptica* had some relationship to the final stages of shipping fever. The isolation of this organism from cattle having other diseases and no lesions of the lungs, and reports from other workers that a small number of normal animals carry *P. boviseptica* in the respiratory tract, would indicate that conditions of feed and exposure might cause this organism to become pathogenic and so produce the symptom complex of shipping fever.

Experiments on the immunization of rabbits and guinea pigs by the use of heat-killed antigens and commercial products give results such as have been reported by Van Es and Martin,[10] who showed that vaccines and bacterins distributed for the immunization of domestic animals against the disease called hemorrhagic septicemia produced no measurable immunity in test animals. Newsom[11] showed that heat-killed Pasteurella vaccines had practically no value in the immunization of rabbits and sheep but that live-culture emulsions had some immunizing value.

The field investigations in Kansas reported elsewhere[12] by one of the authors shows that if there is any immunity produced against shipping fever by the various bacterins and aggressins, it is of very low grade and that the negative period, when the animals are more susceptible to conditions of shipping, is long.

A comparison of the results obtained in the bacteriological examination of material from shipping fever autopsies and the study of the disease on the farms of Kansas would suggest that the causative factor in shipping fever was exposure and improper feeding and care of the animals, and that *P. boviseptica* might have small place in the production of the final disease which caused death in exposed animals. The failure to find this organism in a few cases is probably due to faulty technic but might be due to a probable polybacterial cause of the terminal infection causing death, following the exposures and trails of shipping.

Fermentation studies of thirteen strains of Pasteurella organisms obtained from different sources were made, using salts-sugar-free medium to which 1 cc of a 10 per cent solution of various carbohydrates was added after sterilization. These carbohydrate solutions were inoculated from a 24-hour, salts-broth culture of the organism to be tested.

The results obtained are shown in table VI, in which the four diagnostic carbohydrates, glucose, lactose, maltose and sucrose, are segregated from the remaining eleven carbohydrates.

Two buffalo strains were tested. One (No. 8) was obtained from the Bureau of Animal Industry, Washington, D. C.; the other (No. 4) from a commercial producer of hemorrhagic septicemia products. The equine strain 6, swine strain 7, and ovine

TABLE VI—*Fermentation reactions of Pasteurella organisms*

CARBOHYDRATE	STRAIN OF PASTEURELLA ORGANISM												
	4	6	7	8	17	19	25	28	30	44	48	49	51
Glucose	A	A	A	A	A	A	A	A	A	A	A	A	A
Lactose	—	—	—	—	—	—	—	—	—	—	—	—	—
Maltose	—	—	—	—	—	—	—	—	—	—	—	—	—
Sucrose	A	A	A	A	A	A	A	A	A	A	A	A	A
Arabinose	—	—	—	—	—	—	—	—	—	—	—	—	—
Destrine	—	—	—	—	—	—	—	—	—	—	—	—	—
Galactose	A	A	A	A	A	A	A	A	A	A	A	A	A
Inositol	—	—	—	—	—	—	—	—	—	—	—	—	—
Inulin	—	—	—	—	—	—	—	—	—	—	—	—	—
Levulose	A	A	A	A	A	A	A	A	A	A	A	A	A
Mannite	A	A	A	A	A	A	A	A	A	A	A	A	A
Salicin	—	—	—	—	—	—	—	—	—	—	—	—	—
Sorbitol	—	—	—	—	—	—	—	—	—	—	—	—	—

Buffalo strains, 4, 8; equine, 6; swine, 7; ovine, 25; fowl, 30; Indian cultures from Muktesar, 48, 29; isolated from cases of shipping fever, 17, 19, 28; necrotic laryngitis, 44; bloat, 51.
A = production of acid in 24 to 48 hours; — = no fermentation produced.

strain 25 were obtained from commercial laboratories. The fowl culture 30 was obtained from Doctor Brandley, of the Department of Bacteriology. Cultures 48 and 49 were obtained in 1929 from Doctor Cooper, of Muktesar, India. Cultures 17, 19 and 28 were isolated from outbreaks of shipping fever in Kansas; culture 44 was obtained from a case of calf diphtheria and culture 51 from a case of bloat.

Table VI shows that these thirteen cultures reacted in an identical manner with the thirteen carbohydrates used. Acid was produced in glucose, sucrose, galactose, levulose and mannite.

From this examination it would appear that all the cultures studied correspond to Jones' type 111.[5]

All cultures were motile, gram-negative, short rods and produced small, translucent colonies when grown on salts agar. No hemolysis was produced in horse- or rabbit-blood agar.

Summary

1. *Pasteurella boviseptica* was isolated in 80 per cent of the cases of shipping fever where autopsies were made.

2. In 60 per cent of the 23 cases examined, *P. boviseptica* was associated with either colon-type organisms or with members of the alcaligenes group, usually *A. bronchisepticus*.

3. Immunization of guinea pigs and rabbits by broth and agar cultures showed that *P. boviseptica* had only very slight immunizing powers.

4. A comparison of the agglutination titre produced in immunized rabbits and their survival following test injections showed that there was no direct or constant relationship between the degree of protection and the agglutination titre.

5. A comparison of one virulent culture and an avirulent culture showed that there was no direct relationship between the virulence of a culture and its protective properties. The avirulent culture protected a greater number of rabbits than the virulent culture.

6. Rabbits and guinea pigs varied from 3 to 8 times in their resistance to injections of *P. boviseptica*.

7. Calves were killed by intravenous injections of *P. boviseptica* and colon organisms.

8. Full-fed calves were more susceptible to subcutaneous injections of *P. boviseptica* than calves which were not given hay or grain for 24 hours before an injection.

9. Fermentation tests of 13 strains of *P. boviseptica* show that these organisms produce acid in glucose, sucrose, galactose, levulose and mannite.

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DISCUSSION

DR. J. W. BENNER: There is considerable vagueness about the diagnosis of shipping fever as it occurs in the field. I presume in a study of this kind a certain routine is adopted to make these diagnoses. I am curious to know the principal things upon which the author has based his diagnosis in the field, for these investigations.

DR. SCOTT: The diagnosis of shipping fever was interpreted on rather wide lines. When herds were shipped into Kansas, the buyers were notified, and when disease occurred we visited the farm as soon as possible after the disease occurred. If there was nasal discharge and pneumonia, and the disease could not be diagnosed any other way, we called it shipping fever.

DR. L. W. GOSS: Dr. Scott reports 80 per cent. I presume that not many samples of blood were taken from each herd. Did you find some herds in which there were no Pasteurella organisms present?

Another point of interest is how much attention was paid to the exposure to which these animals were subjected in shipment. Isn't exposure a large factor in the production of disease?

DR. SCOTT: In this paper we have not taken up the question of exposure because Dr. Farley has a paper relative to the incidence of shipping fever in 200 herds examined in the State during the epizoological study of this disease. We did not examine the blood from any sick animals. We simply obtained material from animals which were dead.

DR. A. J. DURANT: What is the mortality rate?

DR. SCOTT: The mortality rate for the whole series (two hundred thousand animals) runs something like this:

In unvaccinated animals we have a mortality of one-half of 1 per cent; in vaccinated animals, treated with vaccines, and so forth, at the farm or at the stockyards, the losses run from 1 to 2 per cent, twice that among non-treated animals, and with animals treated twice, the losses are twice as high as among those treated once.

Seize Rabbits Unfit for Food

Officials of the federal Food and Drug Administration's central district seized, during late November and December, 11 shipments of rabbits which were found to violate the national pure food law. The shipments totaled 996 rabbits, all of which were found upon examination to be partially or largely decomposed and unfit for food. All had been consigned from midwest points to Chicago markets.

The largest single seizure consisted of 250 rabbits, shipped in three wire coops by a produce company in Gorin, Mo., to a Chicago butter firm. They were unfit for food. Officials have recommended seizure of a dozen or more additional consignments and action on these is expected soon. Destruction of the goods has been urged in each case where seizure has been either made or ordered.

THE CONTROL OF BANG'S DISEASE IN RANGE ANIMALS*

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In the preparation of this paper, I have had the aid of a number of veterinarians widely distributed in the cattle-grazing range territory. I wish to take this opportunity to thank them for their very real assistance in furnishing data as to the prevalence of infection and methods of control in their particular localities.

Most of the work on control and eradication of Bang's disease has been done with dairy cattle. Dairy cattle have received the major attention because of their large concentration in areas close to the centers of population, their more detailed supervision, their specialization for milk-production, the serious production losses incurred by the presence of the disease and the desire of men in charge to eliminate these losses. It is needless to state that real progress has been made in establishing methods of eliminating this very costly and insidious malady. Dairymen who have had Bang's disease in their herds are emphatic in their statements that Bang-disease-free herds are much more efficient production units than are herds in which the disease is present. Reliable experimental data are at hand to substantiate this. I mention the work with dairy stock only to remind you that real benefits are derived from maintaining Bang-disease-free herds.

Beef cattle are subject to Bang's disease infection exactly as are dairy cattle. The ravages of the disease are the same, notably lessened calf crop, retained placentas, mastitis, difficult breeding, and sterility. Loss in milk-production is not such an important item as in dairy cattle. Calf loss and sterility are the important consequences to be overcome in beef herds. Another item that is becoming increasingly important is the possibility of undulant fever infection in the men who care for and handle the infected animals. Undulant fever has been rather definitely established as of an occupational nature, occurring most frequently in those persons who have intimate contact with infected animals and carcasses. The possibility of

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human infection warrants mention in any discussion of Bang's disease control.

Cattle-range conditions are to be found in all of the states west of a line extending north and south drawn through the centers of the states of North Dakota, South Dakota, Nebraska, Kansas, Oklahoma and Texas. There is some range or unfenced area east of this line, but by far the greater part of the range country is to the west. I have attempted to make a survey of representative sections of this area to determine the extent of infection and methods of control of Bang's disease in range herds and to compile this information for your consideration and discussion.

Beef herds are classified as strictly range herds, unconfined at any time, and as semi-range herds, collected and held under fence at certain times. Most herds will fall under the latter classification.

Extent of Infection

According to the information furnished me in the preparation of this report, strictly range herds are little troubled with Bang's disease infection. Reports from Montana, Nevada, Washington, Colorado and Wyoming indicate that this disease is apparently not causing serious losses in the strictly range herds. Semi-range herds, however, are reported as becoming seriously infected, and reports from these states and from Texas indicate that Bang's disease is causing measurable losses.

Dr. Edward Records, of Nevada, reports that the presence of Bang's disease in strictly range herds has seldom, if ever, been brought to his attention. Abortion disease occurs to some extent in the semi-range herds, apparently arising in most instances from contact with dairy stock on the ranches during the winter feeding periods. He states:

> On the whole, however, so far as our definite information extends, the loss in this connection does not appear to be excessive or widespread. Occasionally rather severe losses occur in individual herds, but there appears to be a tendency for the disease to subside without artificial methods of control.

Dr. Cecil Elder, of Wyoming, has estimated that infection runs from twenty-five to thirty per cent on an average in Wyoming. He has found the disease in every part of the State and in all grades of cattle.

Dr. W. J. Butler, of Montana, reports:

> Under typical range conditions, ordinarily Bang's disease is not a serious menace. Under semi-range conditions, however, where cattle are ranged in the open in summer and held under pasture during the winter, it is becoming a serious condition.

Dean E. E. Wegner, of Washington, states that there are no strictly range herds in his vicinity. There are perhaps some losses in large herds under fence, but the owners have not asked for assistance. He states that the disease is given scant consideration.

Dr. C. G. Lamb, of Colorado, reports this disease has not yet caused serious losses in Colorado, to the best of his knowledge.

Oregon range herds that have been tested have contained reactors to the agglutination test. The disease apparently is not so prevalent in range stock, however, as in dairy animals.

Dr. W. H. Hendricks, of Utah, has stated that infection has been found in semi-range herds in Utah, but that little control work has been asked for or done.

Dr. N. F. Williams, of Texas, states that little control work has been done in range herds in Texas. Infection is present, however, and the stockmen have done a large amount of vaccinating. No authentic observation of results has been made.

Dr. H. E. Kingman, of Colorado, reports infection in several beef herds, with good results obtained by diagnosis and elimination of infected stock on the basis of the agglutination test.

Control of Bang's Disease

Several experiment stations have been successful in controlling Bang's disease in their beef herds. The Colorado Station successfully eliminated the disease from their beef herd, on the basis of the agglutination test, with five tests over a period of ten and one-half months. The beef herd at Oregon State College was rid of infection in thirteen and one-half months. These herds are classified as semi-range herds. All reactors were immediately removed from the herds, either by slaughter, sale, or segregation.

Tests were not made on any of these herds at exactly regular intervals of thirty or sixty days. Often 120 days elapsed between tests. This is particularly true of the Colorado herd tests. (Colorado Experiment Station bulletin 317.) Quoting:

> The tests were not conducted at regular intervals, but the first three were approximately sixty days apart; the next one was ninety, and the fifth nearly 120.

The Oregon herd was tested at approximately regular sixty-day intervals during 1927 as long as reactors were being found. The tests following 1927 were conducted whenever it was found advisable to move cattle from one pasture to another or to mix two groups of cattle that had been segregated. No reactor

animals were allowed to mingle with the clean animals at any time, or to use the same grounds.

Nevertheless, Bang's disease was eradicated, indicating that it can be accomplished even though testing is more or less irregular. Regardless of this fact, regular thirty- to sixty-day intervals are preferable. Strict sanitary precautions were taken to prevent contamination of the feed and premises of the clean herds. Previous to the application of the agglutination test, a program of sale or isolation of aborting animals had failed materially to retard or eradicate the disease from either of these herds.

Dr. Butler, of Montana, makes the following statement concerning control of Bang's disease in range herds:

> Under strict range conditions the blood testing of range cattle for abortion with the segregation of reactors is impractical. It has been our experience with Bang's disease that affected herds should be tested every thirty days and reactors immediately removed. We have found that if the retests are permitted to go for ninety or one hundred and twenty days, or longer, that we are very apt to get new reactors on every test, showing that we have left a spreader or a latent case of Bang's disease on our previous test. We make it a rule to retest all infected herds every thirty or sixty days and preferably every thirty days.
>
> This you can see is really impossible in range herds. In summer our range cattle are out on forest reserves in the brush and it is simply impossible to gather them even with the best of intentions and the best of men. It is likewise impossible to hold them under fence during the summer as in most instances the fenced lands are irrigated and are used for growing hay.
>
> We have tested quite a number of semi-range herds during the winter months when they were being held in pastures and fed hay. In such herds we removed the reactors and shipped all dry cows.
>
> Under typical range conditions the best method we know of at the present time for controlling this disease is to limit the breeding period to not more than three months and preferably two months; that is, bulls are not to be turned out on the range until July 1 (which is a law of Montana) and are to be gathered and brought in not later than the end of September. In addition to this, we advocate that all dry cows be shipped during the fall shipping season. If these two procedures, limiting the breeding season and shipping dry cows, are followed, the disease will not get much of a start in range cattle.
>
> In semi-range cattle, the same two procedures as adopted for range cattle should be followed, but in addition to this we segregate yearlings and two-year-olds from the older cows and we provide for what we call calving pastures. We advise the owner not to let any cows calve in the pregnant herd, but to have them calve in pastures by themselves or in a pasture that contains cows that already have calved, or steers. In some of these herds where it is possible to follow out the ordinary sanitary procedure and segregation recommended for controlling this disease, we also blood test the herd. We cannot advocate the blood test as general routine in such herds as it is impossible to test them every thirty, or sixty, or ninety days. We do, however, advocate testing all bulls and do not permit any reactor to be turned out on the open range. It has been our experience that in badly infected herds we invariably get an infected bull.
>
> I am quite sure that if the procedures outlined are followed, along with the sanitary precautions of destroying afterbirths and cleaning up infected

grounds, that Bang's disease under range and semi-range conditions can be controlled and in some herds eradicated.

We have used intravenous injections of a one per cent solution of acriflavine with apparently good results. I do not mean by this that acriflavine has cured Bang's disease, but I do unqualifiedly state that in herds where the acriflavine treatment has been used, the number of abortions very materially have been reduced, and retained placenta and mastitis, so commonly a sequel of Bang's disease, practically have been eliminated.

It has been our experience in the case of reacting bulls that are taken out of service for a considerable period (three to six months) that in the majority of cases these positive reactors will become negative.

Dr. Kingman, of Colorado, reports satisfactory results in eradicating Bang's disease from semi-range herds by testing and eliminaton or segregation of reactors.

One herd of approximately 600 grade beef animals was tested. The first test showed 28 per cent infection. The reactors were taken out of the herd and sold for slaughter. The clean herd was moved off the original pasture and placed on new ground. A second test on the negative herd, approximately ninety days later, gave 7 per cent infection. These reactors also were sold for slaughter and the negative herd was again moved to new ground. A third test, approximately sixty days later, gave less than one per cent infection. The reactors were again removed and the herd put in the feed-lot for winter feeding. The owners were highly pleased with the results. The difference in value of these grade animals for breeding or beef was estimated at only $5.00 and the sale of reactors was the logical method of eradication.

A herd of approximately 1200 registered Herefords also was tested. The first test revealed 25 per cent infection. These reactors were removed to a ranch seven miles from the home ranch and which afforded very good isolation. At the end of eighteen months, 8 per cent more reactors had been found and isolated, and at the end of two years, only ¾ of 1 per cent reactors were detected. The abortion rate for the reactor group was 15 per cent and for the negative group less than 2 per cent.

The fact that reactor animals were found in the herd after four and five tests had been made indicates the necessity of repeated testing to eliminate latent infection.

Dr. Kingman states also that the greatest difficulty in controlling the disease in most herds comes from the fact that the owners will not continue to test after active trouble in the herd has subsided. They are lulled into a sense of security and it is difficult to persuade them of the necessity for continued testing.

State Laws and Regulations Concerning Bang's Disease

Some 34 states now have regulations in force concerning Bang's disease, in connection with the interstate shipment of dairy and breeding cattle. Fourteen of these states prohibit entrance of positive reactors to the agglutination or other recognized test. Eighteen states require a negative test. At least one other state (Utah) is preparing to establish regulatory measures. Oklahoma requires a test-chart, showing the reactions, attached to the bill of lading.

Following is a list of states having interstate regulations concerning Bang's disease for dairy and breeding cattle:

Prohibiting entrance of positive reactors except on special permit	*Requiring negative agglutination test*
Alabama	Arkansas
Connecticut	Florida
Idaho	Georgia
Kansas	Illinois
Kentucky	Indiana
Maine	Iowa
Maryland	Louisiana
Massachusetts	Michigan
Mississippi	Minnesota
New Hampshire	New Jersey
North Dakota	North Carolina
South Dakota	Ohio
Oregon	South Carolina
Pennsylvania	Texas
Wyoming	Virginia
	West Virginia
	Wisconsin
	Hawaii

Eight of the seventeen states of the range country (Idaho, Kansas, North Dakota, South Dakota, Oregon, Wyoming and Texas) have regulatory measures.

It is interesting to note the two types of regulatory measures. One type of measure prohibits the introduction of known positive reactors, making no restrictions for interstate shipment of untested cattle. There is a decided laxness in this type of regulation. The other type requires a negative test before admission into the state. This regulatory measure has teeth in it and is sound. It is also interesting to note that only one of the range states (Texas) has this latter regulatory measure. I wish to congratulate Texas on this very definite policy regarding Bang's disease.

Bang's disease is a contagious, infectious disease and as such can be controlled and eradicated by testing to determine the infected animals, and prophylactic sanitation to prevent exposure and contamination of susceptible animals.

The ravages of the disease are somewhat obscure and insidious. Bang's disease does not often destroy the animal, but does impair efficient production to a measurable degree. Much educational work still remains to be done to establish firmly in the minds of the stockmen the real benefits of establishing and maintaining Bang-disease-free herds.

As interest in eradication work increases in the older cattle-breeding states and unloading of infected breeding stock becomes more general, care must be taken to prevent the introduction of these infected animals into the range country. Infection of the range herds is apparently relatively small at the present time. If this be true, then the best time of all to eradicate existing infection and prevent introduction of additional infection is now.

This paper has been prepared for the sole purpose of bringing before you a few facts relating to Bang's disease in range animals, and is presented for your consideration and discussion.

Discussion

Dr. Hadleigh Marsh: I was glad to see there was a paper on the program on the control of abortion in range herds. I think it is the first time I have ever heard it discussed to any extent and the problem is certainly very different from that confronting the dairy men and pure-bred breeders, where most of the work has been done.

Montana has not been in a position to do a very large amount of work yet,—but is attempting to do some work on the control of the disease in semi-range cattle. During the last two years we have done a great deal of testing in range herds, and following them up. We are getting a good start, following up those herds and retesting them. We thing we are going to get some, what you might call, experimental results on this thing, to see what the actual efficiency of that type of work is.

In addition to these tests on about 10,000 range cattle that are being followed up quite actively or accurately at the present time, we hope to follow a few smaller herds a little more closely and have some good information on what we can do. The disease can't be handled as it is in the dairy districts, but we do think we have a good advantage, as mentioned in Dr. McCapes' paper. In dairy herds, calves are being dropped all the time and infectious material is being distributed all around. No matter when the calves are dropped, there are always susceptible cows.

In beef herds, the calves are dropped within a period of two or three months and then there are periods of from thirty to sixty days before the cows are bred again. During the summer, when the cows are out on range, in the semi-range herd, no abortions are occurring. Of course, a part of the cows are pregnant during those summer months, but it is too early for abortions to occur. In general, the chances for infection are certainly minimized under those conditions.

During the winter time, they can have those cattle under control, because each man's cattle are under control and he is feeding them hay. During the summer, when they are on the open range or forest reserve, the amount of infection being spread is at a minimum.

One of the factors I want to emphasize in the semi-range herds is that, in the winter time, conditions seem to be ideal for spread. As a rule the cattle are fed from hay meadows where hay is stacked. It is spread on the ground and the cows are feeding on the ground where excretions are dropped. Those conditions seem to be ideal for spreading disease, but our experience is that

when infection does get into the herd, under those conditions, you may get a high percentage of abortions, but if the herd is handled as it should be in feeding, the percentage is very small.

I have seen in the sand hills of Nebraska, some years ago, where in several large herds, there was a loss of as high as fifty per cent of the calves, and inside of two years they were having practically no loss. We don't know the reason for that. It may occur in a dairy herd, of course, but we think the breeding period is quite a factor.

We advocate shipping all the dry cows in the fall and getting rid of that stuff which may eliminate the spread of the infection.

DR. C. A. CARY: I would like to inject one question. I am not a range man, but they have some range cattle in the South and the range problem is taken up with beef cattle. I don't know whether you range people have considered this. With the dairy cattle, they don't figure on the bull having anything to do with the transmission of this disease unless he is infected. With the number of cattle you have on the range and the number of bulls to cows—I understand you have about one bull for every twenty or thirty cattle on the range—I don't know how it is in the far West, but in some of the range cattle in Alabama, we have some abortions on the range in the summer, and that cow comes in. The bull immediately goes to another cow. Is there any danger of transmission on that basis? I don't know; I am not prepared to say; I am bringing this question up because it is going to be a problem with which we have to contend.

DR. N. F. WILLIAMS: I can't answer Dr. Cary's question specifically, but it is a general understanding among the veterinarians doing range practice that the bull is of very minor consequence in the spread of the disease. We do know some of our range herds have gone through some very trying ordeals. The cattle men were culling at the end of the second year and found out they were putting out cows that had developed immunity. They were doing away with the profitable cows. Now they are going to another extreme and using a live virus in vaccinating their herds as a rule. They are looking, however, for some practical means of handling the situation.

They are sold now on the proposition of testing the bull, because we require that of bulls entering Texas.

I would like to call attention to one case as an example of what may happen if the veterinarian does not meet the full responsibility that devolves on him when he is handling a ranger's herd. We have one pure-bred, white-faced herd in Texas, maintained under range conditions the year round. The owner was ambitious to comply with any regulations that would give those cattle the highest rating. He tested them three times at thirty-day intervals. He tested again at six months, without any reactors, and again at the end of the year without reactors. We let him go for a full year with the understanding that if he tested clean again we would accredit his herd. Between the tests at the end of the first year and the end of the second year, he bought ten nurse cows. He told his veterinarian, "Get me ten nurse cows and put them into my herd safe."

He bought the ten nurse cows and the veterinarian tested them for tuberculosis and put them in the herd. When the herd was tested at the end of the second year, one of the nurse cows reacted. We could not accredit the herd, and the owner said that he was not interested any more, if he could not get his herd accredited, and he dropped the matter.

If the veterinarian had certified those for tuberculosis and contagious abortion, we would have had one herd maintained under range conditions that was happily in control of a man who had the means and could establish it as an abortion-free herd.

I think the situation is clearing some and I really believe the range will offer better facilities for a demonstration of a clean-up on abortion that we are going to get in the dairy herd. We are going to have nature's help out there on the range.

DR. MARSH: In answer to Dr. Cary's question, some years ago I was connected with B. A. I. work on abortion and we did some experiments on that proposition of the bull carrying infection from an infected cow to a clean cow. We were unable to transmit it in that manner. We bred the bull to an

infected cow and within thirty or forty minutes bred a clean heifer, and it was not transmitted.

There is one other point I would like to mention. It does not apply to range conditions any more than anything else, but I notice that some of the regulations in regard to the control of interstate shipments of cattle differ. Some say fifteen days before calving and some say fifteen days after for the tests. That view, it seems to me, is based on the wrong principle. It seems to be the idea that right at calving time, they would not get the reaction. The thing is that the pregnant cow is dangerous when put into a clean herd. I think the disease does not always develop until after the cow has aborted. I believe that a negative reaction on a pregnant cow is not safe. We have a good many cases where cows are negative following an abortion.

DR. C. P. FITCH: What percentage?

DR. MARSH: It is a small percentage, but enough to raise a lot of trouble in a clean herd. That regulation ought to be amended. There is a period right around calving when you don't get a reaction. In these cases the reaction does not appear until after the cow has aborted. I think those regulations are based on the wrong idea.

DR. J. H. WIRTZ: Would it be the same immediately after as before?

DR. MARSH: In some cases you do not get a reaction until thirty or forty days after calving, and you did not have a reaction before. A cow that has reacted at any time during pregnancy, I believe, will react at calving time.

DR. WIRTZ: Some of the medical men, in running the Wassermann test, are up against the same thing in testing during pregnancy. They get negative results in positive individuals, around the time of parturition, and I have not been able to find a man who can say why.

DR. WILLIAMS: Have you some means in mind whereby we could avoid the danger we are meeting under the conditions mentioned?

DR. MARSH: I would not accept a negative test on any pregnant cow. I am not sure these regulations are justified anyway.

DR. WILLIAMS: I do not believe that any of us think we have ideal regulations, but we are doing the best we can.

DR. B. J. KILLHAM: We again have some criticisms of these interstate regulations. Again we are dwelling too much on the exceptions. I don't believe data can be presented to show that the average animal will not react when pregnant or right after dropping a calf. We are talking about the exceptional cow. We know the average infected animal will react any time you test it. We have animals occasionally that won't react while carrying a calf and some within fifteen days of dropping a calf. They are not the rule but the exception.

DR. FITCH: Some of these questions cannot be answered, as Dr. Killham has pointed out, but you men in control work have met exactly the same situations in regard to tuberculosis and progress has been and is being made.

I think I can answer Dr. Marsh by stating that the percentage of animals does not exceed one per cent of the total number of animals that are tested. We have three groups of animals that are being tested today: the negative, the positive, and the suspicious. These animals which Dr. Marsh is talking about and which do occur—don't let's hide that—represent a very small number of animals, but here is the answer. Buy from herds that have been tested and are clean. You would recommend that to your clients in tuberculosis. You should not go into the stock yards and purchase animals to put in a clean herd and have them tested for tuberculosis only. That has been tried too often with too dangerous results.

DR. A. E. CAMERON: Did I understand Dr. Williams to say some range herds had been vaccinated with live vaccine?

DR. WILLIAMS: Yes.

DR. CAMERON: I would think that would be dangerous. You would be spreading it.

DR. WILLIAMS: Doctor, we realize that, but we have no way of controlling it. The cattle men must try that out and get tired of it themselves and then they will support our program.

DR. CARY: Did the veterinarians do that?

DR. WILLIAMS: No—it was done by cow men and cow punchers.

DR. H. E. KINGMAN: As a practitioner who has sneaked into this section on sanitation and hygiene, I want to enter a plea for the practitioner. I hope that the time is coming when this eradication program will be turned back into his hands under the guidance of you research workers, as soon as possible, and I believe it will be as soon as tuberculosis testing has been done.

I hope that the practicing veterinarian in the field will rapidly become capable of taking over this eradication program, and that while the county agents can give great assistance, they will assume a secondary role under the advice of the local and extension veterinarians.

I believe that the greatest single factor in the training of the practitioner is to place in his hands a reliable test, which I believe, as the result of the last five or six years of experience, is rapidly approaching a high degree of perfection, and I believe that is proven when Dr. Fitch says it is the exceptional cow that fails to react, and as that is the case in tuberculosis, as well as in Bang's Disease, then the practitioner, as soon as he has mastered that part is safe with this test.

DR. P. F. MEYER: Why do the state regulations prohibit cattle infected with Bang's disease being shipped into the state when they allow the live culture to be shipped in there and broadcast over the state?

CHAIRMAN CREWE: All regulations in every state prohibit the shipping of cattle infected with contagious, infectious diseases. Some states prohibit the incoming of live organism products and live virus. We permit only the things that pass through our experiment station.

We had some experience with abortion vaccine in the hands of a so-called veterinarian. He inoculated every animal in the herd with it. The result was there was a flame of abortion. This was brought to the attention of the Live Stock Sanitary Board. We have the same requirement regarding hog cholera virus. It can be handled only by licensed, registered veterinarians who are trained to handle it.

DR. KILLHAM: With reference to Dr. Kingman's remarks, I would like to explain that in Michigan we are dealing with the practitioner. We also insist on his being involved. When we do any collecting of blood samples, we do it merely in the way of assisting the local veterinarian. If you study the situation, you will realize that sometimes you can use the county agent to advantage. He can line up the herd without being criticized of being interested financially. The veterinarian might not be able to do that unless he had the full confidence of the people in his community.

DR. C. E. COTTON: I think we, as control men or practitioners, cannot continue to kid ourselves that we can control our vaccines unless we can get the United States government to pass the necessary regulation for the distribution of live viruses. Laboratories are licensed to manufacture these products. In Minnesota they are licensed; they are permitted to move these within the state. We can put up all the protection we want to within our state. We understand, however, that to prosecute a serum company selling virus in our state we wouldn't get to first base. We would have to get to the state in which they are conducting their laboratories and they would beat us in the courts, because of a license from the federal government to move it. We will have to get the federal government to stop the manufacture and moving of living cultures for Bang's disease, and that is the only way to stop it.

CHAIRMAN CREWE: We know that absolutely. We know it is bootlegged in now to some extent, but our law does have a moral effect. The farmer then gets the impression it is a dangerous thing to handle.

Animaldom

Animaldom is the name of the monthly publication of the Pennsylvania Society for the Prevention of Cruelty to Animals. Each issue contains quite a number of interesting anecdotes bearing on humane work with animals. The subscription price is twenty-five cents a year, which just about covers the cost of mailing. Address: 922-24 North Broad Street, Philadelphia, Pa.

THE CARE OF THE EWE BEFORE AND AFTER THE LAMBING PERIOD*

By L. A. HAMMERS, *Clearwater, Kans.*

It is not my purpose to present in this paper anything radically new to the profession. It is a modest subject and probably will not appeal to a veterinarian as it would to the average farmer and sheep-raiser, but to some of you practitioners, to whom sheep-raising is not familiar, it may furnish material for advice to your sheep-raising clients. It is not intended that this article will be of much interest or enlightenment to the shepherd of large ranch herds, but rather just a little common sense advice to veterinarians from districts where small herds and small farms are the vogue.

The first item to consider is the feeding care before breeding. Starting two weeks before the breeding date, put the ewes on alfalfa or clover hay if available or on a good new prairie pasture. Feed one-fourth to one-third pound of linseed meal to each ewe daily. Old, thin ewes should have a grain supplement of one pound of grain per day. This may seem impracticable and have a bit of seeming extravagance about it. Nevertheless, it pays in the future lamb crop.

At the time of breeding, I trust that it is understood that the ewes should be properly docked and if badly infested with burrs these should be removed by clipping or combing, insuring safety to the genital organs of the buck.

The number and quality of the bucks should be well determined and their physical stamina built up to mate that of the ewes. Otherwise the good care of the ewes may be in vain. For every twenty-five to fifty ewes there should be one buck of proper age and stamina. If the bucks are old and slow there should be one to every twenty-five ewes.

The practice of changing from regular, close-cropped pasture to a new location with fresh, uncropped grass, two weeks before breeding, is a good and common practice among sheep-breeders, and certainly adds effectively to the production of twins, and a good average living lamb crop. Grass-grown stubble fields are a common source of new pasture for sheep in my territory and are certainly a good flushing agency.

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It is as essential, when going into the sheep-raising business, to know the chemistry of your soil as it is the balancing of the rations. To have a successful lamb-production the ewes should pasture on land rich in lime and phosphoric acid. This has a great deal to do with disease resistance, even as much as climate and altitude. It also governs, to a great extent, the hardiness of the ewe and her new-born lamb. Lambs from ewes on sandy, washed-out soil are quite often weak, undersized, and of slow growth and a low average livability is obtained. Many a young farmer has tried raising sheep on low, sour, poorly drained land and failed hopelessly, not because of poor care of his flock, but because of the devitalizing influence of the bacterial flora, parasitic infestations, and soil deficiencies.

Avoid Plethora in Ewes

After the breeding season is passed, the ewe requires less careful attention until the lambing period approaches. She should have plenty of range and exercise and good feed, but not an exclusive leguminous diet, or the lambs will be oversized and the ewes too fat, causing them to have difficult deliveries. When the ewes are running on prairie pasture they should be fed a small ration of grain—one-fourth to one pound each, as the condition and age of the ewe would indicate. In the fall, ewes will take sufficient exercise on pasture, but during the winter months exercise should be induced by scattering feed at distant points in the pasture or along fence-lines. In case leguminous feeds are scarce, the linseed meal ration should be increased and a mineral and vitamin supplement supplied, either mixed with the salt in troughs or fed in a ground grain mixture, all the time the object being to increase vitality and govern plethora.

There are some calls for preparturient surgery in the case of ewes, and this is of interest to the veterinarian. Eversion of the vagina is one of the most common, and probably is caused by a costive condition, or from overloading of rumen and bowels. This condition is treated surgically by cleansing thoroughly, replacing the everted portion and closing the vulva by three or more interrupted sutures of linen tape to prevent recurrence, leaving the sutures in place until a normal condition seems evident or until the time of parturition, when they are usually torn loose by the forceful expulsion of the fetus or are removed by the attendant.

Accidental or traumatic abortions are quite common in very fleshy young ewes, and can be prevented by care and good judg-

ment in feeding and housing the heavy ewes. Some things to be prevented are fright by being chased by strange dogs, slipping on sleet-covered ground or ice, jumping logs or high entrances to barns, and rough usage by the attendant. Abortions accompanying an outbreak of hemorrhagic septicemia are embarrassing to the veterinarian, as the bacterin used is quite often blamed by the owner as the cause of the abortions; but if the owner is a reasonable man this can be offset by citing instances of vaccination in healthy herds without a single abortion resulting.

I have had no herd in charge which, to my knowledge,has been afflicted with a contagious abortion. The most annoying feature of the abortions in ewes is that they do not, in a majority of cases, expel the placental membranes before they become fetid and the ewe feverish and off feed. I have learned to give a warning prognosis and use the most cautious procedure in accomplishing the expulsion of the membranes after an abortion. My favorite douche for this condition is a quart of mineral oil to which has been added a dram of bismuth-formic-iodide powder and one-half ounce of powdered charcoal, well mixed by shaking and injected with a horse catheter and syringe or a pressure outfit. I find this gives excellent results although it may have to be repeated in some cases. It is quite the common practice now, at my location, for the owner to bring these cases to the hospital in a truck or trailer for this treatment.

Acidosis or Mineral Deficiency?

"I have a ewe down heavy with lamb. Can you do anything for her?" is one of the common calls during lambing season. I usually find that they have been down several days before the call and that they have gradually gone off feed and become dull and discouraged in countenance. We pronounce it acidosis or a mineral deficiency, or both, for lack of definite knowledge. I leave ferrogen or some other good tonic and a mineral supplement to be given with salt, and usually find that the patient has succumbed after lingering one week to ten days, unless the lambs are born soon and the supportive treatment continued. I said "lambs" were born; for this condition is most often found preceding the birth of twins or triplets. We again urge the use of mineral supplements in pregnant ewes.

We will pass from preparturient to postparturient conditions, leaving parturition, as the subject indicates, to another discussion. Some of the most common conditions encountered after lambing

are eclampsia, postparturient paresis, septic metritis, mastitis, pneumonia, eversion of uterus, agalactia, disclaiming of lambs, retained placental membranes, and anemia.

Eclampsia, as we see it in the ewe, comes on with convulsive movements of the limbs, head and neck, followed by prostration and convulsions, and is quickly cured by injecting calcium gluconate in doses of 50 to 150 cc in each axilla. Milk fever, or postparturient collapse, is recognized by paralysis of the posterion limbs and by the absence or decrease of sensation, from the rump forward, and is cured by calcium gluconate as in eclampsia. Also by the inflation of the udder, and careful protection from inclement weather. Septic metritis is recognized by an elevation of temperature, stiffness of the flank, arched back, straining and anorexia. Cases under my care have best improved by the mineral oil treatment mentioned earlier in this article and the use of bacterins every 48 hours, along with a tonic and laxative.

Mastitis is frequently met with in the ewe but responds readily to treatment of continued hot baths and camphor-menthol liniment with massage, if treatment is started before a stony hardness has developed; I also administer formalin per os two times per day.

Pneumonia a Common Malady

Pneumonia is a common malady following lambing, especially where there has been prolonged decubitus or hemorrhage, and exposure to cold earth or severely cold, damp weather. Pneumonia is best combated by stimulants, such as ethereal camphorated oil (hypodermically) and the beechwood creosote preparations, and a tonic per os.

Agalactia is not an uncommon condition met with in ewes, and might be prevented by proper feeding during pregnancy. Flush feeding and continued hot baths to the udder have been tried but noticeable results have been nil. Dr. Williams mentions an infectious agalactia in sheep and goat herds but we have not encountered this in our practice.

The disclaiming of lambs by the ewe is an annoying circumstance to the care-taker and usually takes time and patience and the tying up of the ewe for a week to ten days, before she will voluntarily submit to nursing the lamb.

Eversion of the uterus in the ewe is treated by careful replacement, as in other species, and is much more easily accomplished

than in the bovine species, as the ewe's posterior extremities can easily be elevated, but the shock of eversion and the consequent replacement is much greater to the sheep than to the cow, and the careful housing and protection of the ewe is important indeed.

Anemia follows lambing, especially in old, poorly fed, parasite-infested ewes. Some will recover sufficiently under tonics and flush feeding to give the lambs a livable amount of milk, but if this condition persists the ewe should be disposed of.

There are conditions of which I have not treated in this paper but it would not be interesting to recite a text-book to so kind an audience. My intention has been to stimulate an active interest in the commonplace care of ewes that you might in turn pass this stimulus to your clients.

Discussion

Dr. E. M. Nighbert: As practitioners, you should know that the sheep business is gradually changing into smaller flocks and on more farms. That means more diseases and more conditions with which you will come in contact. The study of sheep diseases now is an outstanding problem before the veterinary profession and farmers and sheepmen of this country. Of course, I should say that sheep respond to treatment for diseases just as promptly and as satisfactorily, generally speaking, as do other animals.

A few years ago I completed an extensive worm control problem in Silver County, Missouri, where I had 2,000 sheep and lambs under my supervision for nearly five years. Just as Dr. Hammers said, if they are managed right you have very little trouble with them; but some of the trouble, as he explained, did occur where conditions were such that the man could not do all that was necessary.

Let me give you this idea. It may serve some of you younger men. Remember that the old electuary way of treating sheep is a good one. You mix up a sort of syrup, if you want to have the medicine given every few hours or two or three times a day. That is a fine way to do it. Put it on a paddle and wipe it around the mouth and nose. That is a fine way to do it, as the sheep will lick it off, particularly where rams and such as that are off feed and you want to give something to aid digestion.

Of course, I am more interested in parasites. The parasite problem with sheep is gaining ground all the time. They are getting the best of us, there is no question about that. I hope that you people will begin to consider the parasite problem a little more seriously. You are doing well, but you will have to consider the parasite problem a little more seriously than you have in the past. Of course, I do not want to go into a discussion of the treatment of parasites, but we do know something about sheep parasites, particularly the stomach worm. There have been various remedies worked out, but finally they have come back to the classical remedy—copper sulfate. That, used in combination with some other things, will give you fine results. When you consider that copper is one of the essential things of the animal system you will see that in addition to eliminating the stomach worm these lambs will bloom right out. You can continue this treatment. The pastures are permanent now, and we cannot lose the sheep as we used to. They are under fence. I visited all those farms in Silver County, Missouri, and the sheep are still on permanent blue grass pastures. They had all continued the treatment in some way, but two or three had continued the dosage of these lambs every year. The flock was outstanding and in a fine state of health.

PARASITES OF RANCH FOXES AND THEIR TREATMENT*

By KARL B. HANSON, *Saratoga Springs, N. Y.*

Bureau of Biological Survey

U. S. Department of Agriculture

During the past decade or so, fox-farming has shown a steady and rapid growth in this country, particularly in the northern-tier states, where the climate is conducive to the production of good fur. This business has become a permanent and important branch of our live stock industry.

One of the chief phases of fox-farming is the control of disease. In this, because of their special training and experience, veterinarians should take the leading part. Fox-farming offers an excellent field into which veterinarians should extend their practice. Private veterinarians living in fox-farming communities should avail themselves of opportunities to become intimately familiar with the diseases of foxes and to build up a practice with fox-farmers. The veterinarian and the fox-farmer can be of considerable benefit to each other.

Parasitic diseases comprise an important class of the diseases involved in the fox-farming industry.

EXTERNAL PARASITES

The common external parasites of ranch foxes are fleas and ear-mites. Lice and mange are of only occasional occurrence.

Fleas: Fleas are present to a varying degree on all fox-farms, especially during moist and warm weather. They are most prevalent on those ranches where the foxes are kept in small pens or where sanitation of the houses is neglected. The species usually involved is *Ctenocephalus canis.*

Infested animals may or may not scratch themselves. Infestation is best determined by careful examination of the poll, withers, and dorsal cervical regions for the parasites themselves, as well as the characteristic small pin-point-like spots of the skin and specks of flea manure scattered through the fur

A mixture of 1 part of fresh finely ground derris root and 2 parts of flour, talc, or some other carrier, applied as a dust, is a good remedy. Pyrethrum, if fresh, also constitutes a satisfactory

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flea powder, and when it is being rubbed or dusted into the fur, a number of fleas usually become stupefied and fall off the animal. Since a large percentage of these fleas are stupefied only temporarily, they should be promptly destroyed either by burning or by spraying with 3 per cent cresol, kerosene emulsion, or some other effective insecticide. If infestation is heavy, treatment should be repeated in about ten days.

The destruction of the immature fleas is just as important as the destruction of the adult parasites themselves. Since the immature forms tend to collect in the houses, treatment of the foxes should be accompanied by thorough cleansing and disinfection of houses, nest boxes, and chutes. All litter and débris removed from the houses should be promptly burned, and the houses sprayed with 3 per cent cresol or some other effective insecticide.

Fleas seldom become troublesome on fox-farms when the houses are cleaned and disinfected at regular intervals at the times of the year when these parasites are likely to become prevalent.

Lice: Although blue foxes from the islands off the coast of Alaska are commonly infested with the sucking louse, *Linognathus piliferus*, lice are rarely found on red, cross and silver foxes. In the cases in which the writer has encountered sucking lice in blue foxes, these parasites have shown a marked tendency to collect in the region of the eyes, particularly on the upper lids.

When infestation is light and restricted to the eye and head region, local application of 2 per cent cresol is advised. Treatment should be repeated in 16 to 18 days. If infestation is heavy, dipping in luke warm 2 per cent cresol, followed in a minute or two by immersion in luke warm, slightly soapy water, is recommended. When the head is immersed, one hand should be held over the end of the snout in such manner as to prevent the animal from aspirating any of the liquid. Dipping of foxes should be undertaken only on warm days, when the animals can be kept in warm quarters until thoroughly dry. Rubbing down with towels or burlap immediately after dipping is advised.

Ear mange: Otodectes cynotus, the parasite responsible for ear mange in foxes, is present on many fox-farms. Because of the marked tendency of this parasite to spread, the majority of foxes on affected ranches usually are infested.

Mildly infested animals usually show no apparent symptoms, except occasionally shaking the head or pawing and scratching

the ears. In more serious cases the following symptoms may become apparent: drooping of one or both ears, auricular discharge, torticollis, spells of turning in a circle, and convulsions.

In early stages of infestation the deeper portions of the auricular canal, especially the grooves, show a frost-like appearance which is due to the collection of multitudes of the parasites. Soon a scaly-like material collects. In the course of time this material is replaced by a dirty and dark-colored cerumen. As the latter accumulates the number of parasites usually tends to diminish.

An excellent remedy for ear mange is a mixture of 1 part iodoform, 10 parts ether, and 25 parts either of cottonseed oil or of liquid petrolatum. Other satisfactory remedies are a mixture of 1 part naphthol, 3 parts ether, and 10 parts cottonseed oil, and a mixture of 1 part oil of cade and 8 parts cottonseed oil. These remedies are applied to the entire inner surface of the ears by means of pledgets of cotton. Two treatments at an interval of a week to ten days should be given.

The only practical method of controlling ear mange is to eradicate it. This usually will be accomplished if all foxes on the affected ranch are treated at the same time and the houses thoroughly cleaned and disinfected during the interval between the two applications of the remedy.

Mange: Although mange is a relatively common disease of foxes in the wild, only a few outbreaks have occurred on ranches. In most of these cases the disease has had its origin in the introduction of affected foxes from the wild. Mange usually proves a most serious disease once it gains entry to a fox-ranch.

Of six different outbreaks of the disease from which the writer has studied material, five have been due to a species of Sarcoptes and one to a species of Notoedres. Only one isolated case of demodectic mange in a fox has been encountered by the writer thus far. Clinically there is no appreciable difference between notoedric and sarcoptic mange in foxes.

In these animals mange is not readily recognized until after it has reached a relatively advanced stage, because of their long and dense coat of fur. Usually the first thing that attracts attention is that the foxes scratch themselves more than normally and tend to gnaw at certain parts of the body, especially the feet and legs. The affection generally shows up first and most prominently on the hocks, elbows, muzzle, and root of tail. Unless checked by treatment, mange usually spreads over most of the body and eventually results in death.

If an outbreak occurs in winter or late in fall, it generally proves the best policy to destroy all affected foxes as soon as possible. Treatment during cold weather usually proves difficult and unsatisfactory. If the disease occurs in mild or warm weather and conditions permit proper quarantine and isolation of affected animals, attempts at curative treatment may be successful.

Patients to undergo treatment should be kept in isolation quarters and protected from chilling. Clipping the animals is advisable. Dipping the foxes in soluble lime-sulphur dip is the preferred treatment. Treatment should be repeated every 7 to 14 days until recovery. It is essential that reinfestation of patients be controlled by keeping the hospital pens clean and by thoroughly disinfecting them at least every two or three days.

A relatively long quarantine of foxes for mange is advisable. Foxes that have been exposed and those that have recovered from the disease should be kept under quarantine for at least two months.

Infested pens, houses, and other quarters should be left unoccupied for at least two months in warm weather and for a longer period in cold weather. Sometime during the period of vacancy such quarters should be thoroughly sprayed with an effective insecticide or flamed with a kerosene-torch.

Internal Parasites

The common internal parasites of ranch foxes are ascarids, hookworms, lungworms, thread-worms of the bladder, and coccidia. Although various species of tapeworms and flukes are common in foxes in the wild, they are relatively rare on fox-farms. Strange as it may seem, foxes in the wild usually are infested with a much greater variety of internal parasites than are those reared in captivity.

Inasmuch as practically all the common internal parasites of ranch foxes are reproduced by eggs, larvae, or cysts that are voided in certain excretions, usually the feces, the daily picking and safe disposal of droppings is advisable. There should also be good drainage and sufficient sunlight in all pens.

Fish, wild rabbits, and other kinds of meat likely to be infested with immature stages of flukes, and of tapeworms infective to foxes, should be either cooked or subjected to prolonged freezing.

Animals heavily infested with troublesome parasites, particularly lungworms, should not be allowed to remain in the

regular breeding pens; they should be promptly moved to isolation quarters and kept there until they have recovered.

Pens that prove a troublesome source of lungworms and other parasites should be vacated and left unoccupied until they have been treated. Treatments that may be used on such pens are: (1) changing the top 3- or 4-inch layer of soil, (2) flaming the pen thoroughly with a large kerosene-torch, (3) soaking the pen with boiling hot water, or (4) application of certain iodin compounds.

Ascarids: Practically all fox pups become infested with ascarids, usually *Toxocara canis* and occasionally *Toxascaris leonina*. These parasites are common in pups 2 weeks to 5 months of age, but are most troublesome in those 2 to 4 weeks old. Heavy or troublesome infestations are rare in adult foxes.

Heavily infested pups are unthrifty. Their fur is lusterless and feels dry and harsh. Slow growth, pot-belly, emaciation, anemia, restlessness, and digestive disorders are common. Death from obstruction of the intestinal tract with a mass of ascarids frequently occurs in young puppies.

It is advisable to make it the general practice to dose all pups when they are between 17 and 25 days old. Oil of chenopodium, at a dose rate of 0.05 to 0.1 cc per kilogram of body weight and combined with at least ten times its volume of castor oil; and tetrachlorethylene, at a dose rate of 0.2 cc per kilo, are the anthelmintic treatments in most common use. The former usually gives more reliable results than the latter.

Hookworms: Hookworms are present on all fox-farms. *Uncinaria stenocephala* is the species usually involved, but occasionally a few foxes are found infested with *Ancylostoma caninum*, the common hookworm of dogs. Heaviest infestations usually occur in pups 3 to 6 months old, but rarely are hookworms found in pups under 2½ months of age. Adult foxes are commonly infested but seldom harbor heavy infestations.

Poor growth, unthrifty fur, anemia, emaciation, diarrhea, and the passage of large quantities of bloody mucus in the stool are symptoms that may be associated with heavy hookworm infestations.

Whenever losses and heavy hookworm infestations occur together on a fox-farm, the possible presence of a contagious disease should always be given serious consideration before a definite diagnosis is made, even though symptoms and gross lesions strongly indicate that these parasites are the primary cause of sickness.

Occasionally a few pups 3 to 5 months old, rarely adults, require anthelmintic treatment for hookworms.

Tetrachlorethylene seems to be the most satisfactory of known anthelmintics for the treatment of foxes for hookworm. It is given on an empty stomach at a dose rate of .02 cc per kilogram of body weight. Since this drug is attended with some danger of inhalation-intoxication, it is advised that it be given in soft elastic capsules and that care be exercised to prevent accidental breakage of the capsules in administration. Patients should be closely watched for a minute or so after being dosed. Artificial respiration should be immediately performed on those showing signs of collapse.

It is advisable to exercise certain dietary precautions in connection with the use of tetrachlorethylene. One is to restrict fats from the first meal before and after dosing. Feeding the patients a high-calcium diet for a few days before and after treatment appears to be another advisable precaution.

The use of this drug is contra-indicated when there is an outbreak of disease on a ranch.

Lungworms: Due to the extensive losses they cause, lungworms unquestionably are the most important of the internal parasites of ranch foxes.

Eucoleus aerophilus, a fine thread-like worm, is the species usually encountered. A few fox-farms in the northeastern section of the United States also are infested with the lungworm, *Crenosoma decoratum*.

Lungworms generally are more troublesome on shaded ranches or on those on a heavy or a poorly drained soil than on unshaded ranches on a well-drained sandy soil. Old ranches generally are troubled more than new ones. Although pups are considerably more susceptible than adult foxes, the latter frequently become subject to serious infestation.

The predominating symptoms of heavy lungworm infestation consist of a wheeze, rattle, and spells of a deep cough. Other symptoms that may be encountered in very serious cases are an unthrifty condition of the fur, anemia, emaciation, and a lacrymal or nasal discharge.

Lungworm trouble in foxes is a chronic and prolonged disease. A wheeze, rattle, or cough usually is apparent for several weeks before the patient recovers or becomes seriously sick. Except for a few days before death in very serious cases, there usually is no loss of appetite or diminution of activity. It is the chronic and

prolonged nature of the disease that enables one to distinguish cases of lungworm trouble from cases of certain infectious diseases in which the respiratory organs are involved. In the latter the animals show marked depression and usually die within a few days.

The predominating pathological condition encountered in lungworm trouble consists of chronic inflammation of the trachea, brónchi and bronchioles. Bronchopneumonia and alternating areas of atelectasis and vesicular emphysema are common in animals that die of lungworm infestation.

The most successful method of controlling lungworm trouble is to prevent it. On new ranches, and those not troubled with the disease, precaution should be exercised to prevent it getting started. Foxes subject to a wheeze, rattle or cough should be promptly moved to isolation quarters and not returned to the breeding pens until after they have completely recovered. It requires only a short time for heavily infested foxes to contaminate a breeding pen to such an extent that it becomes a troublesome source of serious cases of heavy infestation. Pens in which cases of lungworm trouble develop should be promptly vacated and treated before they are used for the confinement of susceptible animals.

Good success in the treatment of cases of lungworm trouble due to *Eucoleus aerophilus* infestation has been encountered at the United States Fur Animal Experiment Station by the combinaton of two treatments, namely, the confinement of patients in isolation pens with raised, wire-mesh bottoms, and the periodic passage of a tracheal brush at intervals of 1 to 3 weeks. With the exception of only a very few cases in which the condition of the patients had become too serious before treatment was undertaken, recovery from both symptoms and infestation usually has taken place in 2 to 4 months. Occasionally it has taken 5 months to effect a cure. Although a large percentage of patients can be cured merely by confinement in raised, wire-bottom pens, where practically no reinfestation takes place, the use of the tracheal brush appears to hasten and increase the chances of recovery. Although the tracheal brush is of value in the mechanical removal of *Eucoleus aerophilus*, it is of negligible value against *Crenosoma decoratum*. This is because of the tendency of most specimens of the latter parasite to be situated in the air passages below the bifurcation of the trachea.

Thread-worms of the bladder: Capillaria plica, a fine, thread-like worm which infests the urinary bladder and occasionally the pelvis of the kidneys, is present on the majority of fox-farms. This parasite appears to be relatively non-pathogenic. Even heavily infested foxes seldom show symptoms or gross lesions that can be attributed to it. Occasionally a few heavy infestations are associated with either an excess of sediment in the urine or a catarrhal cystitis. There is no known curative treatment. Prevention appears to consist of pen sanitation.

Tapeworms: Despite the fact that tapeworms are very common in foxes from the wild, they are very uncommon in ranch-reared foxes. Infestation is occasionally encountered on a few ranches on which freshly caught fish or wild rabbits are fed raw.

Since no one apparently has investigated the action of the various teniacides on foxes, it is not known what constitutes a safe and effective teniacide for the treatment of these animals.

Flukes: Occasionally a few foxes on some ranches show infestation with the intestinal fluke, *Alaria americana*. This parasite appears to be relatively non-pathogenic. Carbon tetrachlorid apparently is a highly effective anthelmintic against this parasite.[1]

The fluke, *Nanophyetus salmincola*, which causes salmon poisoning, appears to be limited in its distribution to northwestern California, western Oregon, and southwestern Washington. Donham, Simms and Miller[2] have demonstrated that this parasite is highly pathogenic in most carnivores. There is no known curative treatment once symptoms become apparent. On ranches in districts where fish are infested with the immature stages of this parasite, the salmon and trout should either be cooked or subjected to prolonged freezing before being fed to foxes.

Coccidia: Practically all foxes become infested with the coccidium, *Isospora bigeminum*, at sometime or another, particularly between 2 and 5 months of age. Coccidia appear to be relatively nonpathogenic in foxes. Recovery invariably takes place spontaneously in the course of a few weeks. There is no known medicinal treatment to rid foxes of this parasite. Prevention consists of pen sanitation.

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[2]Donham, C. R., Simms, B. T., and Miller, F. W.: So-called salmon poisoning in dogs. Jour. A. V. M. A., lxviii (1926), n. s. 21 (6), pp. 701-715.

DISCUSSION

DR. S. R. JOHNSON: In his excellent paper on parasitic diseases of foxes, Dr. Hanson has not gone into detail. To have done so he probably would have had to write a book. The time at these meetings does not permit of the

detailed treatment which the subject of any parasitic disease really warrants. All I want to do is to enlarge on a few of the points he has brought out.

First is the matter of the veterinarians' relation to the fur industry. It is perhaps true that at the present time many of you practitioners are not located near a fox ranch. However, you don't know when one is going to be established in your community or when you might transfer into a community where there is one already established. If a veterinarian does not take care of the fox man's troubles, they will be taken care of by the empiric or the ranch man, just as you have seen happen in regard to the dog. You will have that same trouble to fight in order to win back this source of revenue.

So I would advise anyone who is located in a community where ranches are already established, or apt to be, to avail himself of any opportunity that he may have to obtain knowledge of this subject.

In addition to the treatment Dr. Hanson advised for fleas, I have found that the addition of from one-half to one per cent sodium fluorid to the pyrethrum is very valuable in destroying the fleas.

As to hookworms, perhaps the most dangerous stage in a fox's life is when it is from two and a half to five months of age. That usually occurs during the warm months of summer. The damage that the parasite does is probably not direct, that is, the parsaite does not kill the fox pup itself, but it does pave the way for bacterial infection.

This bacterial infection is usually limited to the paratyphoid type of organism, hemolytic streptococci or colon bacilli.

Just to emphasize a little further the precautions to be taken with tetrachlorethylene in the treatment of foxes, Dr. Hanson called your attention to calcium deficiency and the danger of fats in the diet, and also diseased conditions. On a great many ranches there may be more or less enteritis of a chronic type. If you are aware of the fact that such a condition exists, you should attempt to correct it before you attempt to treat those animals on a wholesale scale. There is something about the inflammation of the intestinal tract that permits rapid absorption to take place.

In regard to lung worms, the same thing can be said about them as a predisposing factor for bacterial organisms. They undoubtedly irritate the mucosa of the treachea, the bronchi and bronchioles and pave the way for streptococci and colon organisms, causing tracheitis, and in a large number of cases, even pneumonia.

Just another suggestion for the treatment of lung worm as far as the use of wire-bottom pens is concerned. If you have not had experience with these wire-bottom pens, I would caution you or advise you to get a fine-mesh wire, or place a sufficient number of boards so that the animal will be able to travel around reasonably well without the danger of its feet and legs passing through the mesh and in this manner causing considerable irritation and abrasions to occur on the legs. It is just a matter of precaution.

There has often been a question regarding this wire-bottom pen, as to how far it should be off the ground. The main thing is to have it so that the fox cannot come in contact with the ground. If the wire is of reasonably large mesh, the fox's leg can ordinarily pass down through as far as eight or ten inches. He may pick off only a few eggs on his feet, it is true, but if he picks up any, he will lick off his paw and those eggs will get back into his system. Ordinarily I would say the pen should be a foot or fourteen inches off the ground, but that is a matter of personal opinion. Some one else might say less that that.

In closing I would like to ask Dr. Hanson what is known of the life cycle of the bladder worm.

DR. HANSON: The life cycle of *Capillaria plica* apparently has not been fully determined, but it is presumed to be about as follows: The eggs are voided in the urine and undergo an incubation in the soil. This incubation is characterized by the development of an embryo worm which remains within the shell until it is taken in by a suitable host. These embryonated eggs apparently gain access to the host by way of the mouth. After being swallowed, the larvae probably escape from the shell and pass through the intestinal wall.

In some way or another, presumably by way of the bloodstream, the larvae eventually reach the pelvis of the kidney and pass down to the bladder, where they develop to maturity.

DR. C. W. BOWER: I would like to ask Dr. Hanson or some one who deals with foxes if he has had any experience in giving hexylresorcinol for bladder worms. I believe hexylresorcinol has been used recently for ascaris infestation. We know it is a urinary antiseptic given quite often. I am wondering if that property is still in hexylresorcinol when it is eliminated through the urinary tract to a sufficient extent to destroy the parasite.

Question number two pertains to ear mites. I wonder if an active solution of derris root has been used in controlling ear mites. We find it is not so hard to destroy the parasites, but in warm weather their eggs are laid in the fur around the ear and re-infestation is very common.

We have been taking an eight to ten per cent active solution of derris root, swabbing the ear and bathing the whole head and neck almost back to the shoulders, and we have had very gratifying results. This applies also to cats, by the way. Generally one application is sufficient. It has been our experience, if we examine the animal every six to ten days, that we very seldom have to repeat the application.

This is rather new with us but I would like to know if anybody else has tried it. I believe Dr. Perrin told me he uses the same drug in powdered form. He was using it unbeknown to me and I was using the active solution unbeknown to him. I would like to hear discussion on that.

As to the safe dosing of tetrachlorethylene, it is not absolutely one hundred per cent safe. We have had quite a number of losses on our ranch from the administration of tetrachlorethylene in some cases. Fortunately, none of those were administered by myself but out caretaker has lost, I imagine, five or six adult foxes from nothing more than suffocation. They would die as soon as he took them off the table. He may have been a little careless, but the foxes died before anybody could do anything for them.

DR. HANSON: No critical tests apparently have been made of hexlyresorcinol in the treatment of foxes for *Capillaria plica*.

As regards derris root for ear mites, Dr. W. L. Chandler, of Michigan State College, apparently was the first one to introduce this drug as a remedy for ear mites. He proposed its use in a soap solution. This, like the aqueous solution, fails, however, to possess ability to cut and dissolve ear-wax. Ear mange in foxes is commonly associated with an excess of wax in the ears. Microscopic examination reveals that multitudes of live mites and eggs are commonly embedded in this wax. My experience has been that ability to dissolve the wax is a desirable, if not an essential, property of an ear mange remedy in order that it prove effective and practicable.

In the treatment of ear mange in foxes it is best to make a practice of going through and treating all of the foxes on the ranch at one time, and not merely treat those showing definite evidence of infestation. Many lightly infested animals apparently act as reservoirs of infestation. It is also advisable that the treatment be repeated in seven to ten days to kill those mites which may have been accidentally missed by the first treatment, as well as those which may have hatched from eggs subsequent to the first treatment. This double treatment, if thoroughly performed, usually will result in eradication of the parasite.

In regard to inhalation-intoxication, tetrachlorethylene, although less dangerous than carbon tetrachlorid, is also attended with danger of losses from inhalation-intoxication. Immediately after dosing, foxes should be closely watched for signs of collapse until one is assured that the capsule has been successfully swallowed. The secret of success in saving foxes subject to inhalation-intoxication is to administer artificial respiration as soon as possible. Foxes about to collapse usually tremble, show dilation of the pupils, and act groggy before they collapse entirely. Artificial respiration should be performed immediately on such patients. One method is to grasp the animal by the hind legs, whirl it in the air a few times in order to centrifuge the drug from the lungs; then lay the animal on the ground and perform artificial respiration in the usual manner. Make certain that the tongue is not retracted in the mouth so as to occlude the throat while the chest is being pumped. It is surprising

how some foxes apparently dead from inhalation-intoxication can be resuscitated by artificial respiration.

There were also some questions asked in regard to the pens with raised wire bottoms for the confinement of foxes undergoing treatment for lung worms. The wire which appears best for this purpose is one-inch hexagonal mesh, sixteen gauge and galvanized after weaving, woven steel wire cloth. We have had no trouble with sore feet in the use of this wire. The pens are merely a wire-covered table, so as to speak, with a bottomless pen placed on top of it. The bottoms should stand twelve to eighteen inches above ground so as to facilitate getting underneath and cleaning out the feces which fall through the wire. These pens are six feet wide, six feet high, and either twelve or sixteen feet long.

Stabilization in Merchandising

A far-reaching movement to stabilize the marketing of industrial products by means of industry-wide guarantees administered by trade associations and professional societies was revealed in the recent publication, by the American Standards Association, of a report on the first comprehensive survey of the economic importance of certification and labeling activities in the U. S.

The report shows that manufactured commodities valued at over a billion dollars were sold last year by more than 50 industries under some plan of guaranteeing the quality to purchasers. These industries ranged from lumber to mirrors, and from heating systems to drugs.

The report also covers agricultural commodities and dairy products, over four billion dollars worth of which were sold under guarantee last year by means of grading, labeling and certification. Part of this, for example, the classification of eggs as grade A, B, or C eggs, and the classification of milk as certified, grade A, or grade B, is in many cities and states mandatory under the law.

The lumber industry furnishes an outstanding example of the extent to which this new type of marketing has permeated in industry, the report shows. At the present time 35 per cent of all the softwood cut in the United States is marked with a definite grade showing its quality, which the manufacturers guarantee to the purchasers. Nearly 50 per cent of all hardwood is sold under similar guarantees. Over four billion board feet of hardwoods are graded and certified annually.

The American Medical Association certifies to physicians the compliance of certain drugs with the requirements of the United States Pharmacopoeia, including them in its book, "New and Non-Official Remedies." A similar plan aimed to give the ultimate consumer greater security in the purchase of branded foods was recently put into operation.

COUNTY-WIDE ERADICATION OF EQUINE PARASITES*

By JOHN B. BRYANT, *Mount Vernon, Iowa*

This paper opens the discussions on botfly eradication work. The botfly, itself, is in reality an insect. However, the attack is made on the flies while they are in the larval stage and attached to the mucosa of the stomach. Botfly larvae, therefore, are true parasites. The same agent and the same methods used to expel botfly larvae also expel ascarids, hence the subject, "County-Wide Eradication of Equine Parasites."

The parasites concerned in this paper are the botfly larvae and the ascarids. In one of the trade journals is the following and I quote:

> The horse is the most heavily parasitized of all animals. More than 300 different species of parasites infesting the tissues and organs of the horse have been identified. Of these some 300 equine parasites, this essayist has knowledge of only two, the botfly larvae and the ascarids, against which the area plan of eradication has been employed.

I will not attempt to name and identify the various botflies. Eminent parasitologists present may give us authentic information in that regard. While I claim no authority on the biology of these flies, I do maintain that I can speak with certainty regarding the havoc wrought by these pestiferous insects on horse-operated farms. Beginning in June, during July and August, and even into September, these botflies reach that stage in their life cycle when they are depositing their eggs about the nose, chin, shoulders and fore legs of horses in certain districts. Ordinarily steady and true horses will go across the fields throwing their heads, striking with their front feet and lunging about in their fight to avoid the botfly as it lays its eggs and inflicts its punishment.

Various devices, such as nose-baskets, nose-nets, and fly repellents have been employed by farmers in their attempts to protect their teams so that they could work comfortably and efficiently instead of charging about, knocking down the corn and grain, and ruining the religion of the drivers as well as the quality of the field work.

A friend, while touring in South Dakota, in one day, saw two four-horse teams run away while hitched to grain binders.

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

The horses were crazed by the attacks of the nose fly. The two men driving these teams were thrown under the machines and killed. Aside from violent cases such as these, the flies are a nuisance of first magnitude. They are active during the very hot weather, when the horses need all their energy and vitality to do their work and withstand the heat. The added worry and consequent sapping of energy caused by these insects constitute a very serious handicap to horse efficiency. Tractor farmers are not troubled with any such inroads upon their power efficiency and high-pressure tractor salesmen are not slow to take advantage of the opportunity to emphasize this situation. The botfly eradication program is a very splendid conception, indeed, and if eradication can be realized it will be a very worthy enterprise in behalf of horse-farming. It is well, therefore, that this great association should devote time to this comparatively infant undertaking by veterinary practitioners and certain allied interests.

My experience in organization work, on the area basis, is confined to a part of one township, where we treated about 1000 head of horses last winter. Fortunately we have with us a colleague, to appear next on the program, who has had two seasons of experience with county-wide work. I trust he will outline the organization plans for us. I will touch very briefly on organization, as employed in our restricted area. I will then outline my methods of preparation, treatment and after-care of animals, discuss my own observations and conclusions and bring to you a summary of the reports collected by our county agents from owners who submitted their horses to treatment.

The territory was divided into seven districts conforming quite closely to school district lines. Each district has a coöperator who supplied his respective practitioner with a list of owners and the approximate number of horses on each farm. In my work I notified owners with post cards, as to the date and hour that I would be at the farms. I did my work in the forenoons and aimed to treat about 50 head each morning.

Preparation of Animals

Owners were instructed to feed as usual the morning preceding the day of treatment. At noon on this day the animals were to be tied in stalls and all feed removed and the bedding kicked back, so no solids of any kind could be had by the animals. Water was allowed. I emphasized the importance of withholding

all feed from noon until the following morning, pending my arrival. Permitting the animals to run loose in a lot or shed will not do, as they will find some solids to eat and it is my belief that the stomach and small bowel should be completely emptied of solids. My starvation period amounted to about twenty-two hours before capsuling. After capsuling, another two hours of fasting was ordered, making in all a starvation period of about twenty-four hours.

Vermifuge Used

The vermifuge used is carbon disulfid. Previous to last winter, in my worming work I used the liquid, filling a number 10 capsule for mature horses and mules and number 11 capsules for ponies and colts. This past season I used a mass capsule, that is, the carbon disulfid is incorporated in a powdered vehicle and the mass is placed in the capsules. Later I will make a brief comparison of the liquid and mass treatments.

Technic of Administration

This is a very brief description of my operation. The animal is in the full light and the assistant on the right of the horse, with a firm hold on the head stall. The reinforced balling gun is 16 to 18 inches long. The capsule is placed in the gun, cap foremost and then immersed in oil (castor oil preferred), withdrawn and elevated, so the oil runs down over the capsule and thoroughly lubricates it. I then grasp the halter under the nose and elevate the head, pass the gun up the oral cavity to and slightly beyond the base of the tongue and discharge the capsule. The gun is allowed to fall laterally between the molars, and here is the reason I want a heavy gun: It acts as a gag between the molars to prevent crushing the capsule in case the capsule does not lodge in the fauces but comes back into the oral cavity. When sure that the capsule is safely in the fauces I withdraw the gun, watching the left jugular groove for the passage of the capsule down the esophagus.

Not all capsules are observed as they pass. Some horses fidget around and obstruct the view, others swallow very quickly and others have heavy necks and the capsule cannot be seen as it passes. Then there are still others, a very small percentage, that have the esophagus on the right side of the neck, while the left jugular groove is under observation. In quite an extensive use of the stomach-tube I have occasionally observed this atypical anatomical arrangement. It might be well to sound a

warning here. One may become quite adept at passing capsules after doing the work for several days or weeks. This very adeptness may lead to carelessness and if one sends a horse back to his stall before the capsule has safely passed, it may be expelled, and the horse, therefore, is untreated. The object should be to place the accepted dose of carbon disulfid in the properly prepared stomach of every equine in the given area. One animal improperly prepared or one animal that, for any reason, does not receive its dose, becomes a potential menace to the hoped-for 100 per cent results.

After-Care and After-Claps

Owners were instructed to withhold food and water for two hours, then feed as usual. If the animals were accustomed to ranging in a stalk-field, they were turned out. Owners were advised to haul out and spread on the fields the bowel discharges from horses kept in the barn, the object being to expose the larvae and ascarid eggs to killing temperatures. Among the after-claps of the treatment I would first mention colicky symptoms. Quite a percentage of the horses I treated last winter developed mild colics and would not eat for four to eight hours. One very old pony died after treatment. Aside from this no other of my colic cases required attention. While using the liquid-filled capsules I had several of them crushed in the mouths of the horses. Such horses exhibited some discomfort and the buccal membranes bathed in the liquid became very much reddened but there were no bad after-effects from this bungling.

I had three cases where the liquid-filled capsules were lodged in the fauces and retained there until the volatilization blew off the caps. The horses immediately began to cough violently, stagger about, mouth wide open, tongue protruding and the eyes fairly bulging from their sockets. I believe those horses were in real danger of strangulation, due to the powerful anesthetic action of the inhaled gas. It took from three to five minutes for these symptoms to pass off, but I found it advisable to wait fifteen to twenty minutes for the horse to quiet down and for the locally anesthetized structures to regain their sensitiveness before attempting to pass the second capsule. I frequently had mass capsules crushed in the mouth and retained for some time in the fauces with no untoward after-effect.

I have the feeling that the margin of safety is rather narrow in carbon disulfid medication. The fact that animals will some-

times exhibit colicky pains, muscular weakness and even coma (I have never observed the latter) would indicate some disturbance. As the agent volatilizes, there must be some gastric dilatation, and at the point of attachment of the larvae it would seem that the eroded mucous membrane would be very susceptible to the local action of carbon disulfid. In the presence of other gastric disturbances or other weaknesses it would seem that there would be hazards incident to carbon disulfid treatment. However, fatalities are almost negligible when administration has been properly performed.

Mass vs Liquid Capsules

I will now compare the mass and liquid capsules, as regards administration and vermifuge efficiency. In capsuling large numbers of horses some capsules are very likely to be crushed in the mouth or held for some time in the fauces. In either case there is little discomfort to the animal when the mass capsule is involved. In the case of the liquid-filled capsule there is much discomfort to the animal if the capsule is crushed in the mouth. If the liquid is liberated in the fauces there is real danger of inhalation-strangulation. In a very large percentage of animals treated with the liquid-filled capsules, there is leakage enough from the capsule to cause slight to severe coughing, which is not pleasing to the owner.

In my hands the mass capsule was much more readily administered than the liquid-filled capsule. Regarding the comparative vermifuge efficiency of the mass and liquid I wish it distinctly understood that I am expressing my personal views or, I might say, my suspicions, and that I have no experiment data to substantiate these views or suspicions. I reason that liquid carbon disulphid, as such, is more readily volatilized than when it is incorporated in a powdered vehicle. I believe we want rapid volatilization and that we want it completely in the stomach. I fear that some of the mass passes on to the small bowel before it gives up its carbon disulfid gas. I supsect that some of the colics are due to this volatilization in the small bowel. I am quite sure that I had more complaints of colic following the mass treatment than when I was using the liquid treatment. I feel that when the liquid is thrown into the gastric contents or solids, it will liberate its gas more rapidly and completely than will the mass under the same conditions. My personal conclusions regarding the comparative merits of the mass and liquid treatments

give the choice to the mass, as regards administration, and to the liquid, as regards vermifuge efficiency.

I am looking for and I hope some one will supply an explanation for the apparently varied results obtained with this vermifuge treatment. Some owners report the passage of large numbers of ascarids and no bots. Others report large number of bots and no ascarids, and still others report no parasites. On one occasion I searched diligently and unsuccessfully for voided parasites. One treated animal died from injury three weeks after treatment. The owner autopsied this animal and reported the presence of bots still attached. I have the feeling that some owners do not heed instructions regarding fasting, and that some animals are improperly prepared. I do not feel that, in our district, we have attained a proficiency that will accomplish the desired results. The human equation is very much a factor in this work. The owner who unwillingly submits his horses to treatment is careless in the preparation of the animal and delinquent indeed in his condemnation, if striking results are not obtained.

Veterinarians are not out of the equation. The profession set the price for this work at fifty cents per head, which was twenty-five cents less than empirics were getting, and yet in some sections a further cut was made by the profession. Even bidding for the work has been reported. A standard of treatment with high efficiency needs to be evolved and adhered to if success is to be realized and I am of the opinion that that standard will require a fee more nearly approaching the empiric's price than the competitive price that obtained in professional circles in some localities.

Education Needed

Education is needed in this work and it must be of the right sort. Absolute eradication is not to be expected unless full coöperation of all concerned is obtained. This is to be emphasized. It would be better not to treat for an owner than to do it on false premises or uncertain promises. The work will be sufficiently misunderstood and misinterpreted even under the best of circumstances. I cannot refrain from mentioning one man's interpretation of the work. He moved into the treated district in March and called me to come to his farm to vaccinate his horses against the flies. He said he had heard that I had a "vaccination that keeps all flies away from the horses."

The county agent sent out 147 questionnaires to ascertain results. A canvass of the replies, of which there were 39, would

indicate to me that 5 were unfavorable, 2 neutral and 32 favorable. Many report a much better condition of their horses, many report little trouble from the flies, and two report the flies as being worse. I do not think the questionnaires indicate much on which to judge results.

Discussion

Dr. F. H. Kelly: I think that Dr. Bryant gave a very excellent paper. I happen to be the colleague he mentioned who probably started the first work of this kind in Iowa. I told him beforehand that I would give you the organization plan that we carried out.

We started this plan through the Extension Service of the State College, working with our County Agricultural Agent. The first thing that we did was to start the educational plan. We had an extension veterinarian and also an entomologist come from the State College. They had a lantern and a series of slides which educated the farmers to the habits of the nose fly and the various botflies. We have commonly heard the farmers say, when the question is put to them, that they have been advised that the botfly never did any particular harm. They have said that they have had them for years. It is not uncommon to have a farmer tell you that he never had nose flies until he started to raise sugar beets. Those are some of the ridiculous remarks which you hear.

When these men went into a township, which had an active farm bureau organization, everybody was extended an invitation whether they were farm bureau members or not. These lectures are given in the different school districts. There will be a director from each school district present. We then take a blackboard, outline the township, and then divide it into school districts. We call for someone living in each particular school district. Generally we get the school director or somebody else who is rather active. We ask that person to line up all the horses in that particular school district of, say, two miles square. In that way there seems to be no trouble at all in getting a man to line them up. When he gets them lined up, he notifies the local veterinarian, who, when he is ready to do the work, administers the capsule. All that we have to do is to notify this director and he notifies the farmer the day that he expects to be there and about the fee. In that way we get over a township very thoroughly.

During the first year I used the liquid capsule. Results were very gratifying with regard to eliminating bots. Lots of times we did not see the bots. The reason for that was that we administered the capsule during December, or in the early part of January, and the bots were very small and located in the middle of the feces. In fact you have to know where to hunt for them. You are not going to see them lying there like ascarids.

Later I used a mass capsule. I do not believe that we had the results that we had with the liquid capsule. In treating horses of all kinds—mean horses and gentle horses—quite often they bite on the capsule. Being a mass, it would drop out of the mouth and not cause the irritation or the sensation that the liquid would.

I think quite a little of the laity. I generally explain to the farmer that if a capsule bursts in the mouth of a horse, it burns the horse. But as you all know, carbon disulfid merely has the same effect on the horse that a piece of ice would have on your bare back. You stand rigid for a moment until you become accustomed to it. I believe that when the horse breaks the capsule, it does not burn him, but that it either gases him, in a severe case, or causes—not an irritation or a burning of the mouth, but merely this freezing sensation.

Quite often it comes up that the nose fly is the fly that stings the horse. It is my understanding, from various entomologists—and there have been several from Washington and from the State College who come up there to follow out a survey and who tell me and it is also in the United States Department of Agriculture bulletin on botflies—that this fly deposits its egg at the

root of the hair on the lip and it causes a tickling sensation which could be likened very much to the sensation caused by putting a straw in your ear. It just drives you crazy, but it does not sting. At least they have never been able to find a stinger on the fly.

Now, in regard to the various forms of colic. When you are treating a great many horses, say 3,000 or 4,000 head, during December, January and February, it would not be uncommon to lose some horses that would die a natural death if you never saw them during that time. But if you were unfortunate enough to be there on the particular day that the old gray mare was going to die and happened to administer a capsule in the morning, and she died in the afternoon, then the owner would blame the capsule.

I have had fellows say that a man who is a good horseman is generally a fairly good feeder. Horses which have been well fed are hurt a little when kept off feed for eighteen hours. They paw, and every time the barn door is opened they start eating the manger. The minute you have given the capsule and leave the place, very often these good feeders throw in all the feed that the horses have missed for eighteen hours. There is no reason why the horse should not have a digestive disturbance. I always caution them to start feeding the horses gradually in two hours. If a horse has been accustomed to getting six ears of corn and a gallon of oats, I advise cutting the ration in two or giving only one-third.

If there are several attendants around, I always watch every capsule go down, and the minute I give the capsule I have an attendant stand there. (We had a lot of alfalfa hay last year. We do not have it this year, because we did not have the rain.) I have the attendant stand there with a bit of alfalfa. As soon as the capsule is given I let the horse take a nibble of the alfalfa which he takes readily and the capsule will go down where it belongs.

It might be of interest for me to mention something in regard to restraint. When we started in, some of these horses would strike and bite, and some of the horses that never saw a stranger would shy or try to jump over the manger. So we had to work out the easiest and the most efficient way for giving the capsule. Some horses can be given a capsule from in front of the manger, by putting a twitch on them. Others will be handled more easily by leading them out. If they have a bad colt, I quite often bring it up to a post and have a fellow stand there. Then I stand off to the side and give the capsule. I was unfortunate in having a couple of loads of South Dakota range horses come in. There were forty-eight head. They will tell you that you can do this and do that. You know how those horses are which have never seen anybody. In order to make it 100 per cent efficient, you have to capsule every one of them. I would have these fellows run in a dozen of these horses or as many for which we had stall room. We generally threw a rope on the front leg and tied it down to the bottom of the manger. In that way you generally could work in front of the manger and, if he did start to rare, he had to keep one foot on the ground and he did not keep the other one up very long.

It was not uncommon for a man to say that it was worth the price of admission for the administration, just because of the fact that their horses did so much better.

The nose fly, in my mind, is just like this grasshopper condition we read about. If nobody does anything about them, naturally you are going to keep on having more grasshoppers; but if a large group or a certain percentage do something about them, you are naturally going to eliminate some of them if you do not eliminate all of them.

We tried to eliminate our whole county this year. This is my second year at it. I have capsuled over 7,000 head of horses and all of my neighboring veterinarians have done likewise. The first year my neighbor was the only one who capsuled horses, and this year they all did. We have found that it runs rather haphazardly by trying to do too large an area. We would rather take a township and do it absolutely solid than to try to do every township and skip somebody.

The old question came up that the nose fly, of its own volition, does not follow a team more than a half-mile. So, quite often, if one fellow did not treat on one corner of a section, possibly there would be no trouble in the whole section until those horses went over on that side.

Now, in regard to the price. The first year that we did it, when the extension veterinarian and the entomologist came up there, I asked what the price was. The veterinarian said that the year before they had capsuled a few horses and that they were charging 25 cents for horses and 15 cents for colts. Everybody knows that carbon disulfid is used and everybody knows what it costs. I said, at that particular time, "I think that it would be harder to capsule a two-year-old colt than it would be to capsule an old horse; therefore, I think that the price should be the same for all of them."

He asked, "What do you think would be fair?"

I said, "You know that you are going to have a certain amount of hard luck. I think that 50 cents would be O. K." We established the price of 50 cents.

The first year there was quite a bit of publicity given to it. There were a lot of commercial houses—by commercial houses I mean wild cats—who came into our county just on the strength of the work that we did and who were going to build up a big prosperous business on the work that we had tried to do. In our county we call it a project. In other words, if the farmer is willing to line up his own horses, we are willing to treat them at a very nominal fee. That is the impression that we aim to make. We are working in conjunction with the farm bureau. In other words, if there is any cussing out, they will get it instead of the veterinarian. There are two ways to look at that.

So, in our county, before we knew it, the second year these fellows came in from a certain chemical company, went around and sold the capsules. They grabbed off a farm bureau member and gave him half. He would sell the capsule to the farmer for 40 cents and have any veterinarian he wanted to give it. Some of them said they would do it for 15 cents if the farmer bought the capsule. The price varied from 15 to 25 cents. We had a meeting of our county and I suggested to the boys, "In order to shut them out and shut them out quick we can capsule these horses for 40 cents. It will show up our county agent in good shape." We have three right around us who absolutely work for the veterinarian and have him at heart. When a vaccination school comes up he tries to avoid giving it. Those are very embarrassing circumstances. Everybody, in my mind, has to make a living. These county agents, I know, did work in conjunction with our veterinarians. In fact every time our county association has a meeting, we have a little social affair, and we invite the county agent and his wife. We have warned them not to buy these capsules from these wild-cat companies. He wrote a severe letter to all his farm bureau members not to buy this capsule. The result is that John Brown or Dick Smith comes in and says, "Why can't I make half this money as well as the veterinarian?" Here is the farm bureau agent between the veterinarian and the county agent. I said, "All right, we will give these capsules for 40 cents." We were buying them at a nominal price. "That will make it look to the farm bureau as though the veterinarian was back of his county agent and as though the county agent had accomplished something."

I think that when you fellows start capsuling the second year, the 40-cent proposition will be much easier to get than 50 cents.

DR. L. A. MERILLAT: The question before the house is very interesting to me just at this time because Illinois is attempting to follow the program that has been mapped out and carried out in Iowa for the past two years; but as Dr. Hall said in our meeting last summer, this is all pioneer work. In fact, it is very pioneer, this plan of attempting to eradicate worm parasites. This is the first movement in the history of veterinary medicine anywhere in the world to eradicate parasites systematically from domestic animals. Whether Iowa has been doing this for two years or Illinois for one year, as Dr. Hall said, it is still in the pioneer class of veterinary service. It is something that veterinarians should seize upon as one of the outstanding improvements to help animal husbandry in the United States. I believe that it will not stop at driving out a few bots from horses, but it is the beginning of a world-wide attack upon one of the outstanding menaces to animal husbandry.

I think that, inasmuch as the question of price was debated here to such an extent, that should be regarded as one of the least significant parts of this big program. In fact, I have not a great deal of patience with veterinarians who make the matter of price in this thing the major part of the program. Here is

an opportunity for the veterinarian, for the first time since tuberculosis eradication was started, really to become an important factor in helping our animal industry in a large way, and if we can do so and still make a living at it, I think whether we charge 40 cents or 50 cents should not be a very great, important, debatable question. I speak at some length on this because of the fact that it is one of the dominant things that seems to be concerning the Illinois veterinarians today. The important things are those that have been brought out here by Dr. Bryant and by Dr. Kelly, the purely technical things and the things that the association sponsoring the movement has asked the veterinarians to help solve, that is, the question of dosage, the question of how to administer, the question of what drugs to use, in what form to use them, what time of year they should be administered, are all big questions in which we are concerned and for which we should be complimented for having been asked to solve them by the farm bureaus, extension services of the universities, and those who plan these campaigns for us, line up the farmers, give them instruction as to their method of preparation, and then ask the veterinarian to go in and administer the drug.

This, I believe, is one of the very important questions that should be settled —whether the liquid carbon disulfid is better or the solid, or whether something else might not be important.

In regard to the relative value of carbon disulphid in driving out bots, I understand that the time of the year has a great deal to do with it. Dr. Hall recommended to us that the proper way would be to give two treatments, one in December and one later on, in February, and then probably all the bots would be eliminated. This is one of the things for you men, who are doing this work in a large way, to solve: how it should be given, whether with a stomach-tube, in a capsule, as a solid, or as a liquid. Those, as you see, are still debatable questions, inasmuch as such distinguished men as Dr. Bryant and Dr. Kelly are not in accord.

I am going to ask you to call on Dr. Hare, of Kentucky, who has done a lot of work on horses that are worth more than 50 cents apiece. I should like to have him get on the platform, pinch-hit for me, and tell you how to do this job.

DR. FRANK HARE: Mr. Chairman, I appreciate this very subtle compliment that Dr. Merillat just now gave me when he suggested that I tell you how to do it. This area or county eradication plan is something, of course, entirely new to us. We have not approached the problem from just that particular angle. We take up the work at the request of the owner himself, and our methods perhaps of treating animals for parasites are just a little different from what has been described here this morning. I think, perhaps, that that would be the only thing in which you are interested.

In Kentucky we feel that the problem of parasites in horses is perhaps the biggest problem that confronts the breeder of Thoroughbreds. It is analogous to the problems in poultry, hogs, sheep and cattle in other districts where those animals are raised intensively. We have fields there that have been in pasture and on which horses have grazed for fifty or seventy-five years, and those pastures have never been plowed up. Consequently they are thoroughly saturated with parasites. So it constitutes a very serious problem to the horse industry. The breeders themselves have begun to realize that their horses were not doing well, that they were not getting the growth that they should get and that they wanted, and the insurance companies, after paying out several large policies (one of them as high as $50,000 on blood worm infestation), came to the stern realization that, after all, the parasites constituted quite an important economic problem, and they have been energetic in encouraging the owners to treat their horses. I feel that that is about the most satisfactory work that I do. I feel that the horseman himself thinks that he is getting more for his money. He can see results from it, and he does not pay much attention to the fee. That is the least of his worries.

We are dealing with animals that are valued all the way from $500 to $150,000 or $250,000. Consequently we must use drugs that are as safe as possible, but still get the parasites. After all, getting the parasite is the important thing. It is not a question of the safety of drugs. We have to use drugs that get the worms. That is the thing. We all have our ideas perhaps about what

drugs should be used and how they should be administered, but I will give you an idea of what I use.

When I first began worming horses, I used to make microscopic examinations of the feces to determine the presence of parasites. After treating the animal I made another microscopic examination to see what the results were, and it was after making some seventy-five or one hundred such examinations that I decided that my method of procedure was correct, that I was eliminating the parasites practically 100 per cent.

The drugs that I use were not original with me. I simply have taken the work of Dr. Hall and carried out what he has suggested. We use carbon disulfid to eliminate oestrids and bots, and we use oil of chenopodium to get the strongyli. I find that the two drugs are practically 100 per cent efficient for those respective parasites. I have used carbon tetrachlorid, but my results were not satisfactory. I used it on about six or seven head. I got toxic symptoms in one animal that I attributed to the carbon tetrachlorid, and I have never used it since. I know other veterinarians who have had similar results with carbon tetrachlorid. It may have been that the symptoms which appeared after the administration of carbon tetrachlorid were due to something else, but I personally attributed it to the drug.

Some veterinarians have told me that they have had unsatisfactory results from the use of oil of chenopodium, that there seemed to be a marked toxic action, and that the animal would go off feed and stock up in the legs, and that all in all the drug seemed to be quite dangerous.

I believe that the thing that is of very great importance, and perhaps the most important thing, in the use of oil of chenopodium, is to prepare your animal before you dose him. That is important. We use it in a little larger doses than Dr. Hall recommends, but we carefully graduate the dose to the individual. If an animal looks heavily parasitized, instead of increasing the dose of oil of chenopodium I decrease it. I find that in animals heavily parasitized there usually is an entertitis and colitis, and oil of chenopodium will be more toxic and more irritating when a condition like that exists.

We used quite liberal doses of carbon disulfid, and I have never seen any cases of colic following the use of it. However, I have seen them stagger a little bit and sometimes lie down for a few minutes, but it is transitory and I never pay any attention to it. I walk on out of the stall and go on and treat the rest of them.

In regard to preparing our horses, we give them a bran mash tonight, for instance, and take their hay away from them. Tomorrow morning we will give them carbon disulfid. Tomorrow noon we will give them another bran mash and another tomorrow night. The following morning we will give them oil of chenopodium. We usually find that the animal, if treated along about eight or nine o'clock in the morning, will be purging at two or three o'clock in the afternoon. That is quite fast for linseed oil to go through a horse, but if properly prepared I think that that is the way it will happen. I feel that the important thing is to eliminate the oil of chenopodium from the animal's body as rapidly as possible and not allow it to stay in there. If you allow it to stay in there you are going to get toxic symptoms, but if you flush it through rapidly and take out the worms then the animal will not suffer any inconvenience. I give quite liberal doses of linseed oil. I have heard people say that they got super-purgation from linseed oil, but I have not seen it yet in my experience.

We have treated in the last two years about 1500 head of mares, foals, yearlings, stud horses and stallions, twelve and fifteen years old; and I have not seen any cases of super-purgation.

After this dose of linseed oil the animal opens up in a few hours and the bowels move quite freely for about eighteen hours, and then they start checking up. That is the way I like to see them act. I like to see them purge freely for about twelve or eighteen hours. I believe that your results will be more uniformly better if they do purge freely

We use the stomach-tube on foals and aged horses. I have gotten to the point where I use the colt stomach-tube on aged horses. I find frequently that I encounter polypi in the nasal passages and a large tube does not pass very freely, while a small tube will pass. We have to go pretty easy with the Thoroughbred. He is a fighter when he is stirred up, and he will not quit.

We have found two horses in which we were unable to pass the stomach-tube. One of them was a two-year-old stud, and the other was an aged mare. We treat foals in July, and we recommend that they be treated in July. They are about three or four months of age. We find that we eliminate a lot of respiratory troubles by treating foals for oestrids early. As you all know, the oestrid in the horse is very similar to the ascarid in the hog. It passes through the liver and gets into the lung and it creates a little inflammatory condition there, accompanied by cough, pumping, and rise in temperature, discharge from the eyes, and slight discharge from the nose. You will find that a great deal of that is eliminated by treating foals for worms along in July. We treat them again after the first heavy frost in December, and then we may go on and treat them again in early spring, depending on how they look. They gain rapidly and they do well; and all in all the owner is very much pleased with the results that he gets, and we feel that it is a big part of out business.

I have an arrangement with one farmer that if he will allow me to treat his horses when I want to, I will treat all the cases of influenza or these fever cases that occur in his foals free of charge. I have not lost any money on him yet. Last year I had one case. That idea is becoming quite generally spread among the breeders of horses. They are thoroughly sold on the proposition.

About dosage, as I said, we graduate the dose pretty carefully to the individual, but I have given 20 cc of oil of chenopodium and a quart of linseed oil to aged mares heavily in foal, in November, due to foal along in February or March, without any bad results. That would be about a 1050- or 1100-pound mare. That is a little bit larger dose than is ordinarily recommended. We give foals 4 or 5 cc, depending entirely on the size. We also graduate the linseed oil quite carefully. We give a 900-pound yearling about 850 cc of linseed oil and about 15 cc of oil of chenopodium. That would be my dose if the animal appeared to be in pretty good condition, that is, not heavily parasitized.

As far as carbon disulfid is concerned, we give a yearling weighing 900 pounds about 20 to 25 cc. That is close to an ounce. Of course in the aged animals we will give them 30 cc, a full ounce or maybe a little more. With foals we will cut down the carbon disulfid to a little smaller dose and give them 8, 10, or 12 cc, sometimes, depending entirely on the size of the animal. From my experience you can not give anything definite with regard to doses. You have to look over the animal. I go in the stall and size up the animal before I ever pour out the dosage for it.

We put the carbon disulfid in a little glass graduate and cover it with water. Put it in before it vaporizes and the gas bubbles are given off. We see gas bubbles in the water coming off as we pour it down into the stomach-tube.

A very interesting thing occurred one time. I opened a can of carbon disulphid and I used about half of the can. As you all know, it does not pour well out of a can, so I poured it into another bottle, put a cork in the bottle and put it in my car. Sometime after that, I was going to treat an animal and I used this carbon disulfid. As I took out the cork I noticed that there was not that strong pungent odor that you get with fresh carbon disulphid. I treated the animals anyway. I found on microscopic examination that I had not gotten any worms. I went back and treated them. I think that since then Dr. Hall has told us that the principle that destroys the worm parasite is the gas that is given off.

DR. T. H. FERGUSON: Kindly explain a little more fully about the administration of the carbon disulfid through the stomach-tube.

DR. HARE: We attach a funnel to the end of the tube, pour out the dose of carbon disulfid, put the water over it, hold up the end of the funnel and let it drain in. We do not pour out the carbon disulphid and let it stand around. We pour it out just before we use it. I think that you will get better results. There will not be as much gas given off. We may pour a little water in afterwards. We tip the horse's nose up, give him a little time and it drains in.

DR. FERGUSON: If I understand correctly then, you do not attempt to mix them; you just pour the water in on top?

DR. HARE: The carbon disulfid settles to the bottom and the water forms a layer over it. You will see the bubbles coming up through the water.

HISTOLOGICAL STUDIES ON HOG CHOLERA*

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In a number of hog cholera cases it is impossible to establish a diagnosis from the gross anatomical findings. This is especially true in cases which show only part of the typical complex of lesions at autopsy, such as hemorrhages in the lymph-nodes, kidneys, bladder, large intestines and skin. The finding of a method for a quick and reliable postmortem diagnosis would be of great practical value in the control of this disease. Because of the uselessness of methods used for some time heretofore, a thorough histological study seems to offer some promise. Thus far little attention has been given to the histopathology of hog cholera. Our investigations reveal the interesting fact that the hog cholera virus primarily affects the capillaries and also the larger blood-vessels. The lesions in the blood-vessels are represented by proliferation and necrosis, which are found in all organs throughout the body and which seem to be characteristic of this disease. This fundamental finding gives a satisfactory explanation for the hemorrhages and for the necrosis present in various organs in hog cholera.

This report deals with the results of a histological study of the lymph-nodes and kidneys, which macroscopically show the well-known and most characteristic hog cholera lesions (such as swelling, edema and hemorrhages in various degrees, and occasional foci of necrosis in lymph-nodes, and hemorrhages in the kidneys).

MATERIAL

The material consisted of 34 cases in which the lymph-nodes were studied, including cases of from 7 to 32 days duration. Kidney lesions were studied in 29 cases, which were included in the above 34 cases. Twenty of these were laboratory cases and the others were field cases.

LYMPH-NODES

In regard to the lymph-nodes, it is of interest that Nieberle, in Germany, recently pointed out that acute cases of hog cholera

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are not associated with a hemorrhagic lymphadenitis as is generally believed. He is of the opinion that the infiltrating red blood corpuscles in the lymph-node parenchyma are the result of resorption of hemorrhages in other parts of the body by way of the lymph stream, and are not necessarily associated with other lesions in the lymph-nodes.

If this theory is right, the problem of the histological nature of hog cholera would be only partly solved, because it does not offer any information concerning the presence of hemorrhages in other organs. According to this theory, hemorrhagic lymph-nodes should logically occur in hog cholera where hemorrhages exist in the areas drained by lymph-vessels which flow into the lymph-nodes in question, but this is decidedly untrue. Then, red blood corpuscles should be present rather constantly in the peritrabecular sinuses, even in early stages of the process, because they would be carried into the lymph-nodes by way of these sinuses. In early cases, however, the sinuses are free. They are filled only when the erythrocytes are very numerous. Furthermore, considering Nieberle's theory, the question arises as to why lymph-node lesions of the hog cholera type do not occur in other septicemias where there are hemorrhages everywhere in the body (bipolar septicemias, infectious anemia in horses, etc.).

In view of the pronounced inflammatory process and the perivascular hemorrhages in the central nervous system in hog cholera, one's attention is drawn to the blood-vessel system. From the study of our lymph-node material it is quite evident that the hog cholera virus primarily attacks the blood-vessels by way of the blood-stream and leads to more or less pronounced lesions. Working independently on the same type of problem in Germany, Röhrer came to the same conclusion. These blood-vessel lesions must be considered as the source of edema, hemorrhages and progressive perivascular necrosis in the lymph-node parenchyma. The characteristic localization of the hemorrhages in the lymph-node parenchyma is essentially the result of a mechanical distribution of erythrocytes by way of a special lymph-stream in the so-called cell-poor substance of the swine lymph-node, which structurally is different from the lymph-nodes of other mammals. When present there in great numbers, the hemorrhages may enter the sinuses and lead to a crowding and diminution in the amount of lymphoid tissue. Lymph-vessels are free from lesions. Other changes, such as focal hyperplasia, hypoplasia of lymphoid tissue, phagocytosis of erythrocytes and

nuclear fragments by reticulum cells, are the immediate result of the more or less pronounced hemorrhages in the lymph-node parenchyma.

Kidneys

In the kidneys the same fundamental lesions are found in various degrees. Blood-vessel lesions in these organs are likewise responsible for the hemorrhages in the interstitial tissue and in the renal corpuscles. As in the lymph-nodes, there are also focal or diffuse perivascular infiltrations with round cells, a slight edema of the interstitial tissue and foci of necrosis which seem to extend from destroyed capillaries or small blood-vessels. Retrogressive changes of the tubules are evidently due to a combined effect of the virus and of mechanical factors caused by compression from large accumulations of erythrocytes and round cells.

The type of lymph-node and kidney lesions in hog cholera is not directly dependent upon the length of the disease. Furthermore, there are no fundamental differences between the lesions of pure virus hog cholera cases and those in such cases associated with secondarily invading bacteria. However, we have the impression that hemorrhages and foci of necrosis are more pronounced in cases complicated by secondary invaders, such as *B. suipestifer*, and *B. pyosepticus*.

With regard to the practical application of these findings, the study of lymph-nodes and kidneys of a limited number of questionable hog cholera cases shows that the blood-vessel lesions, and lesions resulting from them, may be helpful in establishing a diagnosis of such cases. Further investigations must show whether or not they are strongly diagnostic for this disease.

Summary and Conclusion

In conclusion emphasis should be placed on the fact that injuries of the blood-vessels constitute the primary lesions in the lymph-nodes, kidneys and other organs and that changes in other structures arise as a result of these lesions.

Discussion

Dr. L. W. Goss: We realize that in some diseases where hemorrhages occur the lymph-glands show infiltration. There are some diseases in which the blood coagulates, as in blackleg, at the point of the lesion, but the glands do not show hemorrhagic infiltration. I am wondering if the authors have made a careful study of the afferent vessels of the lymph-glands which show hemorrhagic infiltration, to prove that the lymph-vessels are not carrying blood to the lymph-glands.

DR. CAIN: That was what I had reference to. My reason for showing these lymphatics was to bring out that point. We have had a number of cases where you will have hemorrhages around the blood-vessels and yet no red blood cells in the afferent blood-vessels nor in the peritrabecular sinuses. As a matter of fact, that was true in every case except in those late cases where there were hemorrhages everywhere in the reticulum and the blood goes through the walls of these sinuses.

DR. C. P. FITCH: Were these cases artificially produced or were they cases obtained in a natural outbreak? Were they early stages or were they late stages and, second, have you made any examination of cases of hog cholera where visible lesions were not present? Third, have you made any examination of cases produced by infection with suisepticus or suipestifer?

DR. CAIN: This report includes thirty-four cases, of which twenty were produced artifically. We used four different strains in this work and fourteen of these cases were naturally infected. Of that number some were contacts and some were field cases. Most of these were contact cases. The hogs were inoculated with the virus. As to the duration of these cases, some of them died, and some of them were killed just before they would have died. The course runs from seven up to thirty-two days and most cases run around two weeks or under. Of that number, there were quite a few in which you could see no macroscopic lesions. No inoculations with suisepticus or suipestifer were made.

Methods for Testing Antiseptics and Disinfectants Described

All antiseptics entering into interstate, import or export traffic in the United States are subject to the provisions of the federal Food and Drugs Act, the Insecticide Act, or both, and the government has frequently acted against these products when they have been misbranded. In the enforcement of these acts, confusion has arisen in determining the accuracy of bactericidal and antiseptic label claims because manufacturers do not employ uniform methods of testing their products.

Experience for twenty years has convinced the insecticide control officials of the Food and Drug Administration that certain tests are well suited for judging such products. These tests are described in Circular No. 198-C, "United States Food and Drug Administration Methods of Testing Antiseptics and Disinfectants," just published by the United States Department of Agriculture.

The circular describes several tests, including the Food and Drug Administration (F. D. A.) phenol coefficient method, and the wet and dry filter-paper, the agar-plate, the serum-agar-plate and the agar-cup-plate methods. It is illustrated and includes citations of the literature. A copy of Circular 198-C will be sent free upon request to the U. S. Department of Agriculture, Washington, D. C.

THE HATCHABILITY OF EGGS AND THE LIVABILITY OF CHICKS OF PULLORUM-INFECTED AND NON-INFECTED HENS*

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It would appear that the rôle of pullorum disease, with respect to the fertility and hatchability of the egg and the livability of the chick, has not been the choice of many investigations on this disease. This conclusion is based on the small number of papers which have appeared in the literature on this particular phase of the pullorum disease problem. The problem was considered of sufficient importance to warrant further investigation and a study was undertaken to obtain additional information.

Beaudette, Bushnell and Payne,[1] subsequent to a study on the relation of *Salmonella pullorum* to the hatchability of eggs, concluded that a greater percentage (65.1) of the fertile eggs from non-infected hens hatched, while a lesser percentage (53.58) of the fertile eggs from the infected hens hatched. An even greater difference is recorded in the fertility of the eggs from infected versus non-infected hens. The fertility was 57 per cent in the former and 90.4 per cent in the latter groups. Bulletin 420 of the Wisconsin Agricultural Experiment Station,[2] reporting work by Beach, Strange, Holmes and Halpin, says:

> * * * hens reacting to the agglutination test for bacillary white diarrhea lay as many eggs as hens which are non-reactors, their eggs have equally high hatchability, and the little chicks hatched from those laid by reactor hens are as strong and vigorous, grow as well, and have as low a death rate as little chicks coming from eggs laid by non-reactor hens.

This would imply that pullorum disease is not an important factor in the poultry industry. Investigations were undertaken by Runnells and Van Roekel[3,4] to determine to what extent eggs laid by reacting hens contained *S. pullorum*. They found that from 2.8 to 53.9 per cent of the eggs laid by a group of reacting White Leghorn hens contained the organism. The group average was 14.0 per cent. The average for a group of reacting Rhode Island Red hens was 33.7 per cent.

A biometrical study of the hatchability and chick mortality in a flock of White Leghorn hens is reported by Dunn.[5] He suggests

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that the mortality before (embryonic death) and after hatching is the result of different factors. Some specific factors operating during the stage of embryonic development are said to cause the death of the embryo, while general constitutional weakness is largely responsible for the death of the baby chicks. Dunn states:

> * * * nearly all deaths in the first few weeks are attributable to constitutional causes, or to a constitutional lack of resistance to unfavorable conditions.

Under the caption of "constitutional causes" we are left to ascribe one or more of several specific causes for the death of the chicks and the author eliminates pullorum disease on the basis that the flock from which his data were taken had been tested three times and found free from bacillary white diarrhea. We know, however, that this is not absolute assurance that *S. pullorum* infection has been entirely eliminated. Fecundity studies on a group of reacting and non-reacting hens were made by Asmundson and Biely,[6] with the result that the non-reactors laid more eggs than the reactors.

Procedure

The 209 fowls included in this study were not selected on a basis of breed, type, variety, fecundity, performance or age, except that they were mature. More than half were in the second laying season. Several different breeds were represented and no effort was taken to keep the breeds separate. A separation was made, however, on the basis of their reactions to the agglutination test. In one group were placed all those that gave positive or suspicious reactions, and in the other, only those that were negative to the test. The birds were under close observation for approximately 12 months. Tests were made every 28 days and whenever a bird in the negative group gave a suspicious or positive reaction, it was immediately transferred to the positive group. The eggs were laid in trap nests so that they could be credited to the particular hen producing them. It was planned to keep one male bird for every 12 to 15 females and they were kept in the pens together at all times.

The eggs used for hatching purposes were collected from about November to May, inclusive. Settings were made every seven to ten days during this period. The incubation was done in a large, water-heated, sectional incubator of 2200-egg capacity. A steam-heated room, fitted with specially constructed brooders, was used for brooding and rearing until about 5 to 7 weeks of age. The brooders all had mesh-wire bottoms and they could be cleaned

and disinfected easily. The brooders were further fitted with movable partitions so that the chicks from the particular groups (positive and negative) could be kept separate. On or about the 17th day of incubation, the eggs were placed in bags made of mosquito netting and the chicks hatched out in them. A separate bag was used for each particular hen. Every chick was identified and could be traced to its respective dam.

At the termination of the investigation all the birds were destroyed and a pathological and bacteriological examination made on each.

Results of the Investigation

With the knowledge that the etiological agent in many instances resides in the ovary of a mature hen; in the yolk of laid eggs; in the yolk-sac and tissues of chick embryos and newly hatched chicks, we have then a sequence of conditions fulfilling certain postulates for the transmission of the disease from dam to offspring through the egg. This cycle admitted, it is only reasonable to assume that pullorum disease may play a rôle in connection with fecundity, fertility, hatchability and livability.

The extensive pathological changes occurring in the gonadal tissues of many hens are sufficient evidence to account for a low egg-production; the presence and activity of *S. pullorum* in the yolk may influence fertility; when maturation and subsequent embryonal development occurs, *S. pullorum* often invades the embryo and causes its death; and finally, pullorum disease is the cause of death of many baby chicks.

While the egg-production records on the hens in our investigation are incomplete (covering the second laying season in particular), yet some of the reactor hens produced 115 eggs during the period and others did not produce a single egg. The average for this group was 29.3 eggs. The negative group produced an average of 38 eggs per bird. The range in the negative group was from 1 to 165 eggs. A difference of 8.7 per cent between the reactor and non-reactor groups, while not great, is nevertheless in favor of the non-reacting hens.

All eggs used for hatching purposes were candled on the seventh or eighth day of incubation, to determine their fertility, and those which were infertile were then removed. The results show no difference in the fertility of the eggs from a group of 82 hens that were negative serologically and bacteriologically for pullorum disease when compared with a group of 75 hens which

TABLE I—*Summary of examinations of 209 fowls*

Reactions to Agglutination Test for Pullorum Disease	Birds	S. Pullorum Isolated	Eggs Incubated	Eggs Fertile (%)	Fertile Eggs (%)			Chicks (%) Died From		Raised to Maturity (%)
					Dead Germs	Dead Embryos	Hatched	Pullorum Disease	Other Causes	
Positive	123*	75+	2180	66.6	29.5	44.1	26.2	33.3	33.8	32.8
		48−	1429	74.7	26.5	39.9	33.6	25.6	39.8	34.5
Negative	86	4+	319	65.8	34.2	31.4	34.2	5.5	50.0	44.4
		82−	2963	66.5	30.4	34.9	34.6	3.8	53.7	42.3

*27 of the birds in this group gave reactions which would class them as suspicious.

were positive both serologically and bacteriologically. (See table I.) No attempt was made to examine all these eggs bacteriologically. From time to time eggs from these different groups were cultured and while we sometimes isolated *S. pullorum* from the eggs in the positive group, the eggs examined from the negative group never showed this organism. Many of the eggs in the positive group also were sterile bacteriologically. These findings would give no valid support toward incriminating *S. pullorum* as the specific influencing factor in the infertility of the egg. A second candling was made of all the fertile eggs on the seventeenth or eighteenth day of incubation, to determine which of the germs were still viable and which were not.

The percentage of fertile eggs having dead germs are given in table I. It will be seen that a difference of 8.7 per cent between the positive and negative groups occurred, and again, while not very great, it is in favor of the non-reacting hens. Fifty eggs from each group were examined bacteriologically and it was found that *S. pullorum* was isolated from 13 in the positive and one in the negative group. This of course is not definite proof that pullorum infection plays a large part in the lethality of the germ, but is, we believe, a factor pointing in that direction. The data also show that the number of dead embryos (chicks that are dead in the shell) is greater in the hens which react to the agglutination test than in those that do not react. In 44.1 per cent of the fertile eggs from hens that gave positive serologic reactions to pullorum disease and which were found later to be definitely affected with this disease, dead embryos were found, and also in 39.9 per cent of the fertile eggs from the hens that showed positive agglutinations one or more times during the experiment but from which *S. pullorum* was not isolated. These are to be compared with a group of hens that at no time gave a positive reaction. The percentage of fertile eggs with dead embryos in this group was approximately 33 per cent. Not all of the embryos were examined bacteriologically. However, of 93 from the positive group examined, *S. pullorum* was isolated from 17, but not once was it isolated in 68 examined from the negative group.

Three hundred eight-one chicks were hatched from the 1452 fertile eggs incubated that were produced by the serologically and bacteriologically positive hens. This represents 26.2 per cent hatchability. The hatchability figure on the hens that were positive serologically but negative bacteriologically was 33.6 per cent, on those that were negative serologically but positive

bacteriologically 34.2 per cent, and 34.6 per cent for the group of hens that were negative to all blood tests and from which all bacterial cultures were sterile. These differences, like those for the dead embryos, the dead germs and the fertility, are not great but are indicative of a trend or tendency toward the pullorum-infected hen.

Whereas the results of this investigation have not shown marked differences in fertility and hatchability between reactor and non-reactor hens, there is, however, a great difference in the number of chicks dying from pullorum disease (table I). The mortality from pullorum disease among chicks hatched from double-positive dams was 33.3 per cent; from hens having a positive blood test but which were negative upon cultural examination, 25.6 per cent; from hens that were negative to all blood tests but from which *S. pullorum* was isolated after their death, 5.5 per cent; and only 3.8 per cent of the chicks produced by double-negative hens succumbed to pullorum disease. The differences here are significant and emphasize the necessity of rigorous segregation of reactor hens and the use of eggs from non-reacting birds for hatching purposes.

The negative hens lead in the number of their offspring that were raised to maturity. The number for this group was 43 per 100 chicks hatched as against 34 for the positive group. The mortality figures in both groups on losses of birds from causes other than pullorum disease range from 33 per hundred in the positive group to 53 per hundred in the negative group. "Other causes" include colon infections, infections by fowl typhoid and paratyphoid organisms, fowl cholera, staphylococcic infections, coccidiosis, dietary disturbances and undetermined factors.

Conclusions

1. Hens free from pullorum disease produce more eggs than hens affected with this disease.
2. The data show that eggs from pullorum-diseased hens are no less fertile than those produced by hens free from this disease.
3. The results indicate that at least one of the causes for the arrest of development of the embryo is *S. pullorum*, since the percentage of fertile eggs having dead germs was greater in the reactor hens than in the non-reactor and more of the eggs examined from the former group contained organisms (*S. pullorum*) than those of the latter group.

4. The statistics on the numbers of chicks hatched and the number raised to maturity favor slightly the non-infected hens.

5. The losses from pullorum disease in chicks hatched from infected hens is much greater than from the non-infected ones. From infected hens the loss was 33.3 per cent; from non-infected, 3.8 per cent.

6. The deaths of chicks from causes other than pullorum disease were in excess of those produced by *S. pullorum*. Coccidiosis and colon infections were responsible for the greater number.

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DISCUSSION

DR. L. D. BUSHNELL: I am glad to hear that Dr. Kernkamp agrees more or less with our early findings. I believe that the reports he has made, as compared to ours, can be duplicated most any time. I think I have emphasized on various occasions the great variability of this disease. It is possible to get a group of reactor birds which will give the same hatchability, the same fertility, the same productivity and the same livability as birds which are not reactors. On the other hand, you may get a variation such as Dr. Kernkamp has reported here and you may get variations which are a great deal larger than he has reported.

I don't know why the Wisconsin people get the results which they do. There are certain discrepancies which should be considered before reports of this kind are made. When Dr. Hinshaw was in Kansas he reported on contamination of the test. I am under the impression that a good many discrepancies which have been reported from different laboratories are due to such contamination. There is an organism, which will grow in the presence of phenol, that settles in the bottom of the tube and gives an appearance very similar to a positive agglutination. I do not wish to imply that that is what is complicating the results from other laboratories, but that is one thing that should be considered, and I am still convinced that the results from studies on pullorum disease are very much as Dr. Kernkamp has reported.

DR. H. J. STAFSETH: Dr. Bushnell has made reference to results obtained at the Wisconsin Station. A short time ago, one of my associates was at Wisconsin and called on Doctor Beach, who admitted that some of their claims concerning pullorum disease might be mistaken. We have been working with a flock of hens, varying in age from one to six years, in which we have obtained results similar to those reported from Wisconsin. On the other hand, we know of other well managed flocks in which pullorum disease causes enormous losses, so we do not intend to issue any publications that would lead people to believe that we consider pullorum disease an insignificant factor in poultry husbandry. It is indeed unfortunate that our colleagues in Wisconsin should have seen fit to publish their reports without qualifying remarks.

FOWL-POX VACCINATION AT VARIOUS AGES AND ITS EFFECT UPON NORMAL GROWTH GAINS*

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One of the most noteworthy contributions to our knowledge of poultry disease control in recent years has been the introduction of virus vaccination whereby susceptible fowl may be successfully immunized against pox. Developed by de Blieck and van Heelsbergen,[1,2] the cutaneous method of virus vaccination has been the subject of extensive experimental study. Research workers have conducted studies of various types of vaccines, methods of vaccination and duration of the resultant immunity. The use of a modified virus, as recommended by de Blieck and van Heelsbergen, failed to give satisfactory results in many instances because the attenuated product was incapable of producing an active lesion when applied to the scarified skin. It became recognized that the production of a satisfactory active immunity depended upon inducing a cutaneous reaction in the form of a pox lesion.

In 1927, Johnson[3] reported the results obtained by the use of an unattenuated virus applied to feather follicles. The success of this method of vaccination, as conducted under practical field conditions, is reflected in this author's conclusion that "virus vaccination may be successfuily used in commercial flocks to prevent fowl-pox," and further: "This method produces immunity against experimental as well as natural inoculation." Although the results reported by Johnson clearly indicated a satisfactory method of virus application and uniformity of "takes" by the use of the unattenuated virus, there was some question as to the duration of the immunity established. Further, his results and those of a number of other investigators indicated the hazard attending the use of an unattenuated virus in flocks of lowered vitality.

In the late summer of 1928, the authors, collaborating with members of the Field Division in poultry disease control, began a series of fowl-pox vaccination experiments in commercial flocks. This work was instituted at the request of owners whose

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flocks had shown a yearly incidence of pox infection accompanied by a rather high mortality as well as decreased egg-production. Fortunately, nearly all of the flock-owners coöperated to the fullest extent in this experimental study, permitting the unrestricted movement and handling of birds necessitated by frequent observation of vaccinated and control groups. Mortality sheets and egg-production charts were recorded with the individual pen data.

Similar experiments were conducted on less mature birds during the summer of 1929 and to a lesser extent during 1930. The results obtained during these three years of experimentation involving the vaccination of thousands of birds were not unlike those reported by many other observers. These results may be summarized by stating that the judicious use of a potent pox virus on healthy susceptible birds will successfully immunize them against natural infection with fowl-pox. The immunity induced by a successful vaccination take was found to endure for a period of two years as indicated by failure of a virus of known virulence to produce a visible lesion at the point of inoculation.

Since the beneficial effects of fowl-pox vaccination depend upon the production of an active immunity, it is only natural to assume that the localized lesion is accompanied by a general systemic reaction. Whether or not this systemic reaction results in definite injury depends upon the vitality of the bird. In many instances the disastrous results following the use of virus vaccination may be attributed entirely to erroneous judgment regarding the general health of the flock. These errors, which have occasionally resulted in a relatively high mortality, have caused some poultrymen to question an otherwise beneficial product.

From the experiment data collected from our observation in the field and those of many other investigators, the question as to when to vaccinate is a pertinent one. At what period in the development of the bird is the reaction incident to vaccination least apt to disrupt its physiological balance. From an economic standpoint the vaccination of mature pullets with an unattenuated fowl-pox virus is to be discouraged because of its effect in lowering or inhibiting egg-production for a variable period. Such vaccination of mature pullets is superior only to exposure to natural infection by virtue of the fact that the raction usually remains localized and the flock infection runs a less protracted course. It would appear, therefore, that vaccination against fowl-pox should be done during the growing period of the bird.

To date various recommendations have been made relative to the proper age at which birds should be vaccinated. Johnson[4] states:

> It is not advisable to vaccinate under three months of age except under unusual circumstances.

This recommendation is based upon the author's contention that the fowls probably show a less marked systemic effect after that age. In considering the effect of chicken-pox vaccination on healthy pullets, Beach[5] points out that young fowls should be vaccinated before egg-production has started and cautions against the vaccination of birds not in normal health. Pyle[6] comments upon the effect of cutaneous vaccination on body weight as follows:

> The vaccine does not cause a retardation in the gain in weight following its administration to birds 80 days of age or older. Following its administration to birds 68 days of age there is a slight retardation in weight gains.

Unfortunately the author did not continue to observe the weight gains beyond the stage of active infection.

Recognizing the need of additional experiment data pertaining to this question of the most desirable age period for fowl-pox vaccination, a study was conducted to determine the effect of fowl-pox vaccination at various ages on normal development. It was our belief that the debilitating effect, if any, of the systemic reaction incident to such vaccination would be reflected in the growth gains. While the tabulated growth gains of the various vaccinated and control groups serve as the basic study, we were interested also in the correlation of the extent and character of the reaction on the induced immunity. Data were collected also on the relative merits of the feather follicle and "stick" methods of vaccination by a comparison of the growth gains, vaccination takes and resultant immunity of birds so vaccinated.

PROCEDURE

In order that the experiment data might be applicable to the American and Mediterranean breeds, both were represented in our selection of Barred Plymouth Rocks and Single Comb White Leghorns. These two breeds were selected further because they dominate their respective classes in the poultry population of Pennsylvania. The experiment stock was the progeny of birds comprising the disease-free unit maintained for the purpose of supplying disease-free birds for experimentation. In passing, it may be of interest to record that the birds and their progeny of this unit have remained free of an infectious disease for a period

of four years. No precautions have been observed other than practical isolation and care in the addition of breeding stock.

Experiment 1. Barred Plymouth Rocks. Feather-follicle method of vaccination: The Barred Rock chicks used in this experiment were obtained from a group hatched April 1, 1931. When 30 days of age (May 1, 1931), two groups of five chicks each were selected on the basis of body weight. In selecting these two groups, one for vaccination and the other for control purposes, we endeavored to have the sex distribution and weight totals of each group as nearly equal as possible. The group of five chicks to be vaccinated was removed to house 1 of the disease unit, while the control group of five chicks was maintained in separate quarters in the disease-free unit. Although entirely separate, each group was maintained under the same housing conditions and received like rations and general care. This was done in an effort to equalize those factors in the two groups normally affecting growth and development.

The vaccine used throughout the experiment was an unattenuated virus. This strain of virus had been obtained from a flock in which fowl-pox had run a rather protracted course. By repeated inoculation, its virulence was increased to the point where it regularly produced well-defined vesicles in four days and the formation of scabs seven days after inoculation of the comb or wattle tissue. The virus material collected on the sixth day after inoculation was desiccated in dry air at 37.5° C. for 48 hours. The stock virus was stored in powdered form at 6° C. The vaccine as prepared during this experiment represented a suspension of 8 mgm. of virus per cubic centimeter of 50 per cent glycerin in distilled water. The potency of the stock virus was checked at regular intervals in conjunction with other experiment work pertaining to virus studies.

At thirty days of age, the Barred Rock chicks showed feather development in the region of the thigh. The vaccine was applied to six feather follicles. In all instances where the feather-follicle infection method was used, care was taken to confine the virus application to six follicles. This precaution was taken in order that an accurate comparison of the vaccination takes could be made and thus denote the correlation, if any, between the development of the local reaction and weight gains. Observations of the local reaction began on the seventh day following inoculation and were continued at weekly intervals until healing was complete. On the fourteenth day after vaccination, the chicks

were removed to house 2, where they were housed for an additional fourteen days during which period the vaccination scab had entirely healed. Twenty-eight days following vaccination, the chicks were removed to house 3, where they had access to range.

We view this housing rotation of vaccinated birds as important, in that it minimized the danger of natural infection of birds whose original vaccination takes may have been questioned, particularly when the "stick" method was used. A similar rotation system of housing of the control birds was followed with the idea of maintaining both groups under like conditions. On May 15, 1931, when the reserve group of Barred Rock chicks had reached the age of 44 days, two groups of five chicks each were again removed on the basis of body weight and placed on experiment. This procedure was repeated at two-week intervals until September 4, 1931, when the final group (No. 10) was vaccinated at the age of 156 days. Every bird under experiment, whether vaccinated or control, was weighed at seven-day intervals and the weight recorded in grams.

TABLE I—*Mortality record of birds in experiment 1*

BIRD	VACCINATED GROUP NO.	VACCINATION DATE	CONTROL GROUP NO.	DATE OF DEATH	FOLLICLE INOCULATION	DIAGNOSIS
61	1	5– 1–31		10– 5–31	Healed	Sarcomatosis
71			1	6–20–31		Heat stroke
2	2	5–15–31		11– 2–31	Healed	Exhaustion and injury due to fighting
34			2	6–20–31		Heat stroke
39			2	6–20–31		Heat stroke
72			2	6–20–31		Heat stroke
24			3	6–20–31		Heat stroke
86	4	6–12–31		6–20–31	Active	Heat stroke
124	4	6–12–31		6–20–31	Active	Heat stroke
7	5	6–26–31		11–22–31	Healed	Leucemia
174			6	10– 9–31		Leucemia
164	7	7–24–31		10–24–31	Healed	Exhaustion and injury due to fighting
Y19	8	8– 7–31		9– 8–31	Healed	Sarcomatosis
30	9	8–21–31		10–14–31	Healed	Pneumonia

Deaths in vaccinated groups	8
Deaths in control groups	6
Deaths attributed to vaccination	0

Experiment 2. Single Comb White Leghorn chicks. Feather-follicle method of vaccination: The procedure of this experiment was essentially the same as that followed in experiment 1. Five birds each for the vaccinated and control groups were selected

from reserve at two-week intervals. The experiment began on May 29, 1931, when the chicks were 35 days old. The last group (No. 10) were 161 days old when vaccinated on October 2, 1931. Recording of experiment data pertaining to the reaction incident to vaccination as well as the body weight made were as in experiment 1.

Experiment 3. Single Comb White Leghorn. Stick method of vaccination: The object of this experiment was to aid in determining whether the method of vaccination has had any marked influence upon the growth gains. Should such an influence be noted, it would indicate that the indirect effect of the local rather than the systemic reaction has a bearing upon the health of the bird, as evidenced by a deviation from normal weight gains. A direct comparison of the systemic effect, if any, of the two methods may be made between the data of experiments 2 and 3, for the reason that the chicks of both experiments were vaccinated at the same age and the individual group weight totals were approximately the same. The control groups of experiment 2 served the same purpose for experiment 3.

TABLE II—*Mortality record of birds in experiments 2 and 3*

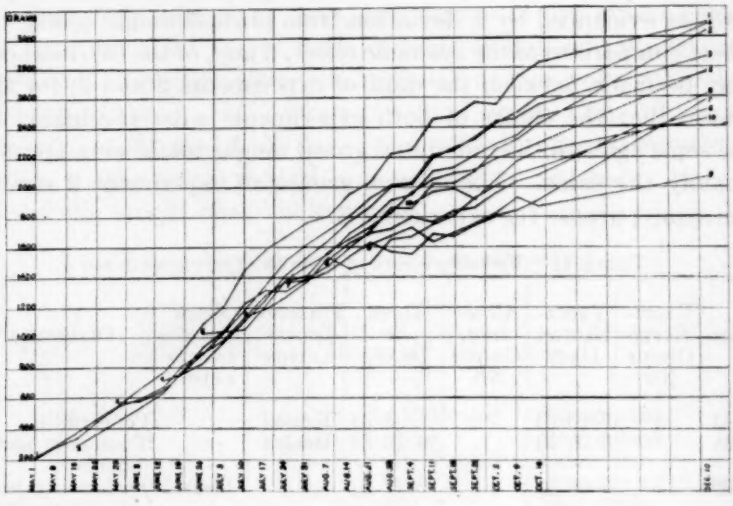
BIRD	VACCINATED GROUP NO.	VACCINATION DATE	CONTROL GROUP NO.	DATE OF DEATH	FOLLICLE INOCULATION	STICK METHOD INOCULATION	DIAGNOSIS
242	1	5–29–31		7– 5–31	Healed		Pericarditis
258	1	5–29–31		*6–19–31	Healed		Traumatic peritonitis
236	1	5–29–31		6–17–31		Healed	Perforating ulcer of proventriculus
232			1	6–19–31			Enteritis
R 2			1	6–20–31			Heat stroke
235	2	6–12–31		7– 1–31		Healed	Heat stroke
348	3	6–26–31		7– 3–31		Active	Mechanical injury
355			3	8–17–31			Exhaustion and injury due to fighting
405	4	7–10–31		7–22–31	Active		Note 1
206			4	8–27–31			Rupture of liver

Deaths in feather-follicle vaccinated groups 3
Deaths in "stick" method vaccinated groups 3
Deaths in control groups 4
*Killed.
Note 1. The cause of death of bird 405 is questionable. It may have been due to vaccination shock.

The chicks were vaccinated with the aid of an improvised tattoo device prepared by permitting four phonograph needles to extend 1/16 of an inch from a cork, at the base of which an

absorbent cotton pad was kept saturated with virus. The medial surface of the patagium served as the point of inoculation. This site was chosen because of its relative freedom from feather follicles. For a direct comparison as to the intensity, extent and duration of the vaccination take, observations of the cutaneous reaction were made on the same dates as those of experiment 2.

On December 3, 1931, each vaccinated group of birds in experiments 1, 2 and 3 was divided into two lots for the purpose of determining the immunity of the individual birds. The birds of one lot were inoculated by means of the feather-follicle method with a virus of known potency. The other lot was exposed to



GRAPH 1—Growth curves of the ten groups of Barred Plymouth Rocks vaccinated by means of the feather-follicle infection method. The dotted line represents the average mean weight gains of the control groups.

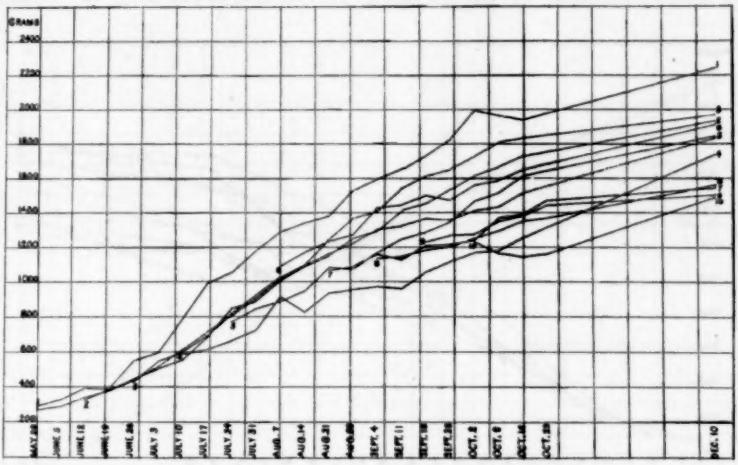
natural infection by cohabitation with three birds which had been vaccinated on the comb and wattles and presented typical epitheliomata.

Observations on the individual birds of both lots were made on the seventh, fourteenth and eighteenth days following exposure. In no instance was there an indication of reaction to either method of exposure. While there may be some criticism of the fact that the test of immunity was made too soon, such tests were conducted at a time when the normal incidence of fowl-pox is at its height under field conditions. The additional observation that the immunity present was sufficient to protect against

artificial infection with an unattenuated virus indicates a durable immunity.

Experiment Data

As indicated in the brief description of the method of conducting these studies, the collected data comprise: (a) body weight in grams of vaccinated and control birds at seven-day intervals; (b) notations relative to the intensity, extent and duration of the local reaction recorded on the seventh, fourteenth and twenty-first days after vaccination; (c) mortality records, including bacteriological, pathological and parasitological examination results; and (d) duration of immunity.

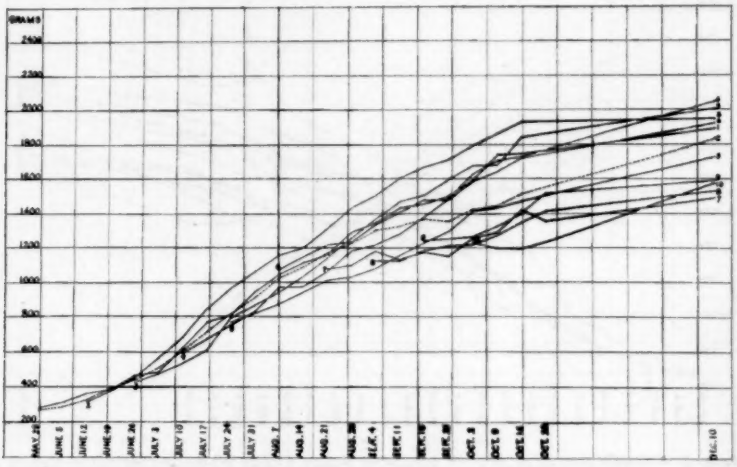


Graph 2—Growth curves of the ten groups of Single Comb White Leghorns vaccinated by means of the feather-follicle infection method. The dotted line represents the average mean weight gains of the control groups.

(a) The data pertaining to the growth gains are presented in graph form to conserve space and to aid in the direct comparison of the weights of the various vaccinated groups and their relationship to the normal curve of the unvaccinated controls. The weights recorded on the graphs represent the group average for that particular date. The actual number of birds, less than five, representing each group on any given date may be obtained by referring to the mortality charts. The growth curve of the control groups, as indicated by the dotted line in each graph, represents the average mean weight of all control birds on experiment per given date. For example (graph 1), the average mean weight of the controls on May 1, 1931, represents the average of five birds, while that of May 29, that of 15 birds. We feel

that this condensed representation of the average mean weight of all controls is justified by our observation that the average weight of the various control groups per given experiment was only slightly affected by the housing rotation previously referred to. Graph 4 presents the average mean growth curve of the vaccinated as compared with the control groups of Barred Rocks. Graph 5 summarizes the combined data of graphs 2 and 3 and serves to compare the average mean growth gains of the feather-follicle and stick-vaccinated groups of birds with the norm curve of the non-vaccinated control Single Comb White Leghorns.

(b) The uniformity in the intensity, extent and duration of the local reaction at the point of virus inoculation was such as

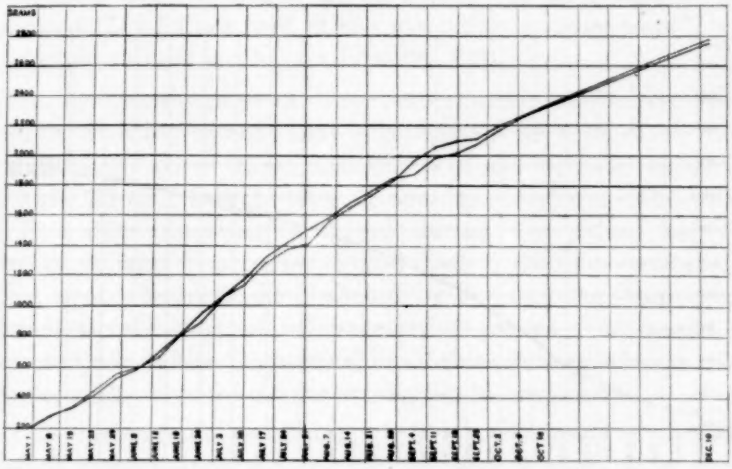


GRAPH 3—Growth curves of the ten groups of Single Comb White Leghorns vaccinated by means of the "stick" infection method. The dotted line represents the average mean weight gains of the control groups.

to preclude the necessity of a detailed presentation. This uniformity of local reactions was most marked where the feather-follicle infection method was used. Observations on the seventh day showed scab development confined to individual follicles, accompanied by either a localized or confluent swelling. On the fourteenth day, the scabs were usually confluent, swelling had subsided and healing was progressing in a normal manner. In the younger chicks healing was often complete when observed on the fourteenth day. Although each vaccinated bird showed a local cutaneous reaction at the point of inoculation, that induced by the "stick" method was rather erratic of development. Not infrequently the presence of a local reaction was seriously

questioned at the 7-day observation, but would be well defined on the fourteenth day. In no instance was the local reaction induced by the stick method of virus application so severe or extensive as that of the feather-follicle method.

(c) While the development of pox and diphtheritic lesions subsequent to inoculation may be attributed to fowl-pox vaccination, our observations in controlled field experiments have shown that the mortality in many instances is due to an independent factor and would have occurred irrespective of whether or not such birds were vaccinated. Consideration of the mortality rate of these experimental groups is particularly important in order to denote the possible correlation between the harmful



GRAPH 4—Growth curves representing the average mean weight gains of the ten groups of feather-follicle vaccinated Barred Plymouth Rocks as compared with the control groups (dotted line).

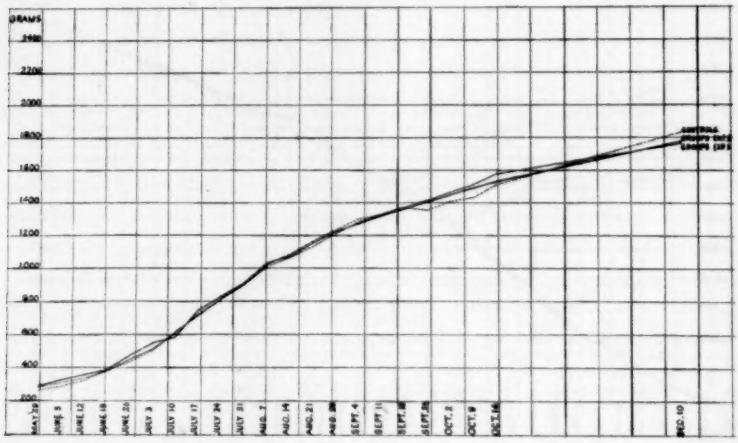
effects of vaccination at the various ages and the death-rate. As an aid to such an interpretation, we have included the vaccination date and a notation relative to the presence or absence of a local reaction at the point of inoculation. The diagnosis briefly given constitutes our opinion as to the immediate cause of death. These diagnoses are based upon bacteriological, pathological and parasitological examinations.

DISCUSSION

Since the objective of fowl-pox vaccination is the production of an active immunity sufficient to protect against natural infection, the question of immunity and its duration is of primary

importance. If the immunity induced by vaccination at different ages is variable and transient, the question of proper age for vaccination would necessarily be based upon the measure of induced immunity. The fact that all of the vaccinated birds of these experiments were immune to not only natural but artificial exposure to a pox virus of known potency, leaves us free to judge the merits of vaccination at the various ages wholly by its effect on the physical condition of the bird.

A study of graph 1, presenting the growth curves of the 10 groups of birds vaccinated by means of the feather-follicle method, will reveal that groups 1, 2, 3 and 4, vaccinated when 30, 44, 58 and 72 days old, respectively, show very slight if any deviation from the normal growth curve, with weight gains in



GRAPH 5—Growth curves representing the average mean weight gains of the feather-follicle and "stick" infection methods as compared with the control groups.

excess of any other group. Group 5, vaccinated at the age of 86 days, showed a virtual inhibition of weight gains until fourteen days after vaccination, when there was a sharp upward trend and a continuation of normal gains.

Beginning with group 6, vaccinated at the age of 100 days, and continuing with groups 7, 8, 9 and 10, we find a distinct break in the normal growth curve. The normal growth and development of these groups prior to vaccination is indicated by the fact that the body weights closely approach the norm control growth curve on the date of vaccination. It will be noted that this break in the normal weight gains of groups 6, 7 and 8 occurs at the termination of the fourth week after vaccination, while

that of groups 9 and 10 shows a loss of weight after the second week. Since other factors remain the same, this regularity of weight loss and attendant physical depression must be attributed to the systemic reaction to vaccination.

In mature pullets this systemic reaction is manifested in part by a lowering or temporary inhibition of egg-production. It would appear from these observations that the systemic effect of vaccination is negligible in birds between the age of one and three months. Further, that the inoculation of the birds older than three months is accompanied by a distinct post-vaccination shock which may be a predisposing factor to continued impaired vitality. While not materially affecting the values of the experiment data, since it was a factor common to all groups, it will be noted that 50 per cent of the mortality in experiment 1 was attributed to heat stroke, on June 20, 1931.

During the progress of these experiments there were three periods of rather intense heat accompanied by a relatively high humidity. Two of these high-temperature periods, occurring on June 20 and July 1, were of one-day duration. The third, of four days duration, occurred during the period between August 28 and September 4 and had a distinct devitalizing effect upon the birds, as is shown by the uniform drop in the normal weight gain of all vaccinated groups. As would be expected, the White Leghorns showed this effect to a less marked degree, with only two deaths attributed to heat stroke.

The observations pertaining to the growth gains of the vaccinated Barred Rock groups of experiment 1 apply with minor exceptions to the vaccinated White Leghorns of experiments 2 and 3. A study of graphs 2 and 3 will reveal that groups 1, 2 and 3, vaccinated by either the follicle or stick method, at the age of 35, 49 and 63 days, respectively, fail to show any evidence of a systemic reaction sufficient to affect normal development.

Group 4 of experiment 2, vaccinated at the age of 77 days, showed subnormal weight gains for four weeks following vaccination, when there was a distinct break with very little gain in weight for the next five weeks, after which the group began a favorable recovery. Whether the post-vaccination behavior of this group can be wholly attributed to vaccination shock is questionable, although such a possibility is suggested by group 4 of experiment 3. This group, representing birds of the same age and weight, shows a temporary inhibition of weight gains

between the fourth and fifth weeks following vaccination with the stick method.

Group 5 of experiments 2 and 3, vaccinated at the age of 91 days, begins to show a slight inhibition in the normal weight gains in the fifth week after vaccination, but this depressant effect is only temporary and should receive slight consideration.

Group 6, vaccinated on August 7, at the age of 105 days, shows a sharp decrease in normal weight gains beginning the fifth week following the use of the feather-follicle method. This decrease in weight gains in the corresponding group of experiment 3 is marked by only a temporary inhibition during the third week. The depressant effect of the systemic reaction incident to vaccination, while less marked in groups 5 and 6, becomes pronounced and nearly identical in groups 7, 8, 9 and 10 of either experiment.

The continued low level of the weight gains of these latter groups in experiments 2 and 3, and to a lesser extent in experiment 1, would indicate that the severity of the systemic reaction increases with age, at least until maturity. A reaction sufficiently severe to inhibit normal weight gains may serve as a serious detrimental factor to future health, particularly in those birds whose normal reserve has been depleted by disease or parasitism. This observation has been substantiated by the incidence of post-vaccination losses in flocks which were moderately parasitized or those in the incubative stage of disease.

Correlating these experimental results with the potential hazards of vaccination during the middle and latter part of the growing period, when birds are passing through the so-called critical coccidiosis age, we feel that vaccination of birds between 30 and 90 days of age is to be recommended. Birds vaccinated during this period of development continue normal weight gains and health. The immunity induced is at least sufficient to protect the birds against artificial infection with, and natural exposure to, fowl-pox virus during the late fall and early winter months when the incidence of this disease is greatest. The mortality which can be honestly attributed to vaccination during this period is virtually nil. Field observations indicate that the mortality increases in flocks vaccinated at a more mature age, due largely to the combined effect of parasitism, disease and vaccination shock.

A comparison of the weight gains of the vaccinated groups of experiment 2 with those of experiment 3, summarized in graph 5,

shows virtually no differences attributable to the feather-follicle or stick method of vaccination. These results agree with the observations reported by Stafseth.[7] If the instrument employed for the stick method is such as to assure the positive inoculation of virus, this method is to be recommended because of the uniformity in size of the local lesion and for conservation of virus used. One of the common abuses of the feather-follicle method is the application of an excess amount of virus to an excess number of follicles.

Conclusions

1. Vaccination of birds between 30 and 90 days of age is recommended. The systemic reaction incident to vaccination during this period does not appreciably affect normal growth and development.

2. Vaccination of birds between 90 and 120 days of age produces inhibition in normal weight gains. In normal birds this period of inhibition is transient and exerts no visible influence on future normal development.

3. Vaccination of birds older than 120 days results in a distinct post-vaccination shock. Under certain conditions this systemic reaction may be a predisposing factor to impaired health.

4. In birds 30 days of age or older, a successful vaccination take at the point of inoculation will produce an adequate protective immunity against natural infection with fowl-pox.

Acknowledgment

We wish to express our appreciation to Drs. R. O. Biltz, R. L. Elsea and C. W. Miller, who coöperated in the field experiments referred to in this paper. These observations were of invaluable aid in serving as a basis for this and other studies pertaining to fowl-pox vaccination.

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THE OCCURRENCE OF COOPERIA ONCOPHORA AND NEMATODIRUS HELVETIANUS IN CALVES*

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The veterinary literature contains very few reports of intestinal strongylosis among cattle. One would be led to infer that this phase of parasitism has not caused much loss and hence is not of much economic importance.

History: A range bunch of cattle composed of calves, steers, and cows were being wintered in a hay meadow on a ranch in northwestern Montana. In addition to the pasture they were being fed alfalfa hay. Fifteen out of approximately 500 calves (short yearlings) were noticeably sick and had been isolated into a hospital bunch. The two worst cases died. After a change of feed the remaining 13 calves gradually recovered.

Symptoms: The foreman in charge of the cattle expressed the belief that the calves had not grown out properly. From this we would gather the idea that the condition had been gradually developing for some time. The general appearance of emaciation, a hump in the back, roughened hair, dull sunken eyes, and a thin, profuse, fetid, and more or less bloody diarrhea were the only noticeable symptoms.

Lesions and parasites: One dead calf showed a hemorrhagic rectal mucosa. Unconcentrated smears contained a few coccidia. The unconcentrated feces showed Nematodirus eggs and coccidia.

From a second calf the unconcentrated scrapings of the small intestines revealed many large and small coccidia. A massive infestation of small strongyles was found in the duodenum and abomasum, while the cecum, lower small intestine, and rectum were practically free of parasites. Concentrated films of compo-

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site fecal samples from affected calves gathered off the ground showed a good many coccidia and some nematode eggs.

The Zoölogical Division of the Bureau of Animal Industry in Washington, D. C., identified the parasites as *Cooperia oncophora* and *Nematodirus helvetianus.*

Diagnosis: The primary cause of this loss is uncertain because of the complicating presence of the coccidia. The symptoms were indistinguishable from coccidiosis as observed in calves and yearlings. Yet, the massive infestation of *Cooperia oncophora* and *Nematodirus helvetianus* precludes this diagnosis. It is a question just how much of a factor the coccidia were and also whether the worms or the coccidia were the primary cause.

The only justifiable conclusion is that one might easily make a diagnosis of coccidiosis from the symptoms and lesions had not the entire intestinal tract been carefully examined before reaching a decision. Certainly sufficient numbers of strongyles were present to cause pathological alterations and visible symptoms. Whether this would have occurred in the absence of the coccidia cannot be definitely stated.

Treatment: Drenching with copper sulfate solution and a change of feed from alfalfa to native hay, with the addition of oats or cottonseed cake, was recommended. An uneventful recovery followed this change of feed. The hospital bunch improved so rapidly following the change of feed that the copper sulfate treatment was not attempted.

CALCIUM DEFICIENCY IN ECLAMPSIA

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Some interesting facts will be noted in the records of three cases of eclampsia in bitches, taken at random from the Small-Animal Hospital. The results of the use of calcium chlorid intravenously are particularly gratifying in comparison with the age-old narcotic treatment, often resulting in death.

Case I

Subject: Poodle, mother of six three-week-old puppies.

History: Diet consisted of meat, bread and vegetables. Milk was absent. Puppies were always hungry. The attack came on rather suddenly, when the animal was presented at the hospital.

Symptoms: Tonoclonic spasms, loss of motor control, rapid respiration; temperature, 108.2° F.; mammary glands, dry.

Diagnosis: Eclampsia.

Treatment: Ten grains of calcium chlorid in one ounce of water was administered intravenously (external saphenous).

Results: In one minute the animal could stand, the spasms subsided, and in 45 minutes the temperature was checked and found to be 100.8° F., and the animal active and normal in all apparent respects. The case was discharged the next day. Calcium lactate, twenty grains three times a day, per os, was prescribed and instructions given to supplement the diet with milk.

Case 2

Subject: Poodle, 18 months old, mother of five two-week-old puppies.

History: Diet consisted of meat and vegetables. Milk was absent. The animal developed illness suddenly. Puppies were always ravenously hungry.

Symptoms: Exactly the same as in case 1, except that the temperature was 107.4° F

Diagnosis: Eclampsia

Treatment: Ten grains of calcium chlorid in 10 cc of water was administered intravenously.

Results: The injection was made slowly into the external saphenous vein. The spasms subsided by the time the injection was complete. The approximate time was one minute.

The animal was discharged immediately, and calcium sulphid prescribed, one grain to be given three times a day and the diet supplemented with milk. The animal was reported in excellent condition two weeks later.

Case 3

Subject: Collie, 1½ years old, mother of seven three-week-old puppies.

History: Diet consisted of Ken-L-Ration and dog biscuits. Milk was absent. The animal appeared normal that morning, but became uneasy at 5 p. m. and refused her meal. Puppies were always ravenously hungry.

Symptoms: Admitted to the hospital at 8 p. m. Languid; temperature, 102.6° F.; incoördinated gait; mammary glands, completely dry.

Diagnosis: Approaching eclampsia.

Treatment: Ten grains of calcium chlorid in 10 cc of water was administered intravenously. Milk of magnesia, 4 drams, and calcium lactate, 20 grains, administered per os.

Results: The animal was discharged three days later, very active; temperature, normal; mammary glands in high state of secretion. The owner was instructed to supplement the diet with milk and was given syrup of calcium lacto-phosphate to administer per os.

Experimental

It was decided to infuse a solution of sodium citrate intravenously, which will inactivate the calcium ion, to observe its effects. (Petersen and Hewitt,[1] University of Minnesota, injected a 20 per cent solution of sodium citrate into cattle and produced symptoms simulating parturent paresis, or milk fever. Their findings were confirmed by Dr. R. S. Amadon, of the University of Pennsylvania.) A male dog was selected for the experiment, to eliminate the sex factor in true eclampsia.

Subject: German Shepherd, male, age 18 months.

Condition: Blood and urine analysis, normal. Temperature, 102° F.

Procedure: The external saphenous vein was used for the site of injection. A 15 per cent solution of sodium citrate was prepared and infused very slowly so that six ounces of the solution was utilized over a period of 2½ hours, the exact time for the experiment.

Ten minutes after beginning the infusion, symptoms of incoördination developed and progressed in double that time to complete loss of motor control. About the same time, rapid respiration began and lasted throughout the experiment. Tonoclonic spasms of the muscles of the limbs, eye and temporal region were well evident. There was a gradual rise of temperature which registered 108.2° F. at the close of the 2½-hour period.

Upon discontinuing the injection of citrate, the latter was rather rapidly eliminated, so that automatic recovery took place in one-half hour. Of course in true eclampsia the calcium is below the normal level, while in the above experiment it was present but merely inactivated.

Conclusions: (1) All three cases were on diets deficient in milk, one of the most available sources of calcium. (2) Mammary

glands were dry in all instances. (3) Administration of calcium brought immediate recovery. (4) Because of the handicap and danger of intravenous administration in practice, calcium gluconate could be used as treatment for eclampsia in plate of calcium chlorid.

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NITROBENZENE POISONING IN THE DOG

By BRUCE KESTER *and* T. W. JOHNSON

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On October 15, 1931, a young Schipperke bitch was brought to the office of T. W. J. Examination showed the following symptoms: rapid, weak pulse of 180; increased but shallow respirations; normal temperature; pupils dilated; profuse salivation, which lasted for 24 hours; abdomen "tucked"; persistent constipation prevailed and the patient lay in a lateral recumbent position, with the two uppermost legs held at right angles to the

FIG. 1 (left). Position assumed by dog between attacks. Note the position of the two uppermost legs.

FIG. 2 (right). One of the positions assumed during an attack. Note the abduction of the upper fore leg. The litter mate is under the influence of a sedative.

body (Fig. 1). Sudden excitement would produce muscular spasms of short duration. During a spasm the uppermost fore leg was always abducted (fig. 2). At other times the bitch would throw herself to the opposite side or backward. Occasionally she would partly gain a sternal position, with the head swaying from side to side.

History: The following history was obtained from the owner: All five dogs were kept in the house most of the time but were allowed the run of the back yard occasionally. However, they never left the premises. All dogs received the same food, which

usually consisted of table scraps and some meat. Their evening meal was a vegetable stew, consisting of lima beans, celery and carrots. Two or three hours after this meal, while the two young bitches were playing in the room, one was noticed to pick up one hind leg in a peculiar manner. Then she raised a front leg in an unnatural manner. Repeating these movements a couple of times, the bitch fell to the floor and was unable to regain her feet.

Purgatives were given without effect. Digitalis and strychnin were administered but the latter aggravated the muscular spasms and was discontinued.

With a symptom complex as described, the authors were at a loss as to the etiological factor. Botulinus poisoning was suspected.

On October 16 (24 hours later), the litter mate became ill, showing the same symptoms. The owner stated that the second bitch had refused feed during the day but otherwise was apparently normal. However, he recalled that he had had some old ground bonemeal fertilizer left from the preceding year and that he had scattered this around the rose bushes a day or so before the first bitch became ill. He remembered that the two young bitches had eaten some of this. Due to the fact that on the third day the three older dogs had not developed symptoms, the vegetable stew was eliminated as the source of the trouble and the ground bonemeal fertilizer suspected. A sample was sent to the Department of Bacteriology, Ohio State Uuiversity. The report was negative for *Cl. botulinus*, both bacteriologically and by animal inoculation.

On October 20, the owner, after receiving an unfavorable prognosis, wished the dogs destroyed. At this time the symptoms were the same as when first observed, with the exception of the absence of salivation. We were yet at a loss to understand what might have been the etiological factor producing this symptom complex.

A definite clue was reached one month later, on November 15, when the old bitch was brought to the office showing the same symptoms as the two young bitches, with the exception that she supported herself in a sternal position with the fore legs abducted. The owner stated that, since the first two cases, the food had been changed and the two remaining dogs had received the same rations. Therefore, we eliminated the food as the source of the trouble. The owner, an art designer, then recalled that he had

used a paint deodorizer as a flea repellent. He had observed that no flies were ever present in a room in which the deodorizer was used. He had used it in the form of a spray the past two years, with no bad results.

Knowing that it was a good fly repellent, he had used it for fleas under the following conditions: He estimated that he had placed about 40 ounces upon the ground floor of the cellar. A board platform was placed over this area. This was covered with straw and was used by the dogs as a bed. There was a store-box upon the bed, which had been used by the old bitch when she whelped. It contained an opening by which the dogs gained entrance, also one or two small cracks. With a brush he painted the inside of the box with the deodorizer and placed straw within for a bed. Only the two young bitches slept in the box, as they would not allow the old dogs to enter. These slept upon the bed outside. This accounts for the fact that only the two young bitches became ill in the first place. It required 24 hours for the first bitch and 48 hours for the second to develop symptoms after sleeping in the box. After the young bitches became ill, the old dogs were not allowed in the cellar until one month later, when the old bitch was allowed to sleep in the box. She slept there two nights and was found ill the following morning.

Experimental: It remained only to test the drug under similar conditions. On November 23, a box was prepared as described, the inside painted with seven ounces of the deodorizer, and then the bottom covered with straw for a bed. A black and tan hound pup was obtained and at noon was placed within; the opening was partly closed to prevent his escape. At the beginning of the experiment, his pulse, respirations and temperature were normal. In the evening, he was fed and allowed to play for a couple of hours. At 7 p. m., his pulse was 135, full and strong; respirations and temperature, normal. He was again confined in the box for the night. The following morning, his pulse was 88 and weaker; respirations and temperature, normal. From this time on, he was allowed to use the box at will. It was observed that he had slept in the box a greater part of the day. In the evening his condition was the same as observed in the morning. At 7 p. m. about three cubic centimeters of the deodorizer was applied to the surface of the body along the region of the back.

The following morning no difference could be observed in the dog. At noon he ate heartily and played. He was observed again

at 4 p. m. Vomitus was present upon the floor. Examination of the dog showed the following symptoms: a weak pulse of 86, dilatation of the pupils, a characteristic downward jerking of the eyeballs, increased full respirations, normal temperature, profuse salivation and muscular weakness. When first observed, the dog was able to stand but walked with an unsteady gait. While walking towards one of us, he would suddenly turn completely over sidewise from a standing position. He would regain his feet but soon was attacked again, this time falling upon his sternum, with head arched backward and with fore legs extended and spread apart, hind legs flexed. From this position he threw himself from one side to the other. The attack was of short duration and the dog was again able to regain his feet. In the course of several minutes the last two symptoms described were again alternated.

Within 45 minutes, the dog was unable to arise but was able to support himself in a sternal position. The head was held up but swayed slightly from side to side. The right fore and hind legs were slightly abducted. In the meantime, it was learned through the drug company from which the drug had been purchased, that it was nitrobenzene or nitrobenzol (in trade, oil of mirbane). It is produced when concentrated nitric acid acts upon benzene. It is a yellowish oily liquid, having the odor of bitter almonds, and is used in art, shoe dye, in the manufacture of aniline, is employed as a scent for soap and it has been learned that it is being used alone, in vacuum, as a spray for moths or in combination as "fly tox" by some manufacturers. The latter is of interest to veterinary medicine only in that the product alone might be sprayed upon the animal.

In two cases of human poisoning, Loeb and Fitz[1] found a very striking reduction in the oxygen-carrying power of the blood. With this thought in mind, oxygen was injected under the skin at 5 p. m. From this time on, the dog lay rather quiet, in a sternal position. By 7:30, the jerking of the eyeballs was less prominent but mild attacks were evident by a slight drop and extension of the head and abduction of the legs. At 8 p. m., oxygen was again injected under the skin. At 10 p. m., the symptoms were the same as observed at 7:30. He was not examined again until 6 a. m., when the dog was found in a lateral recumbent position. He was unable to raise his head and all legs were in motion. This being Thanksgiving Day, we were unable to obtain oxygen and the animal was destroyed. Further

experiments with special reference to treatment would seem advisable. After the death of the dog, the postmortem findings were essentially negative, except a chocolate-colored appearance of the blood.

Discussion: In all probability the poisoning resulted from both inhalation and absorption of the drug. It would seem that the symptoms of nitrobenzene poisoning are due, in part, to the action of the poison on the nerve centers and in part to its destructive effect upon the blood. It is of interest to note that a sufficient amount of the drug remained in the boards of the box to produce poisoning in the old bitch one month later. Nitrobenzene poisoning of domestic animals is unusual, if not unknown. Although unusual in humans, a number of cases have been reported. Stifel[2] reports a series of cases resulting from absorption from the use of shoe dye.

Acknowledgment: The authors desire to thank Dr. W. A. Starin, of the Department of Bacteriology, Ohio State University, for the examination of the material.

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World-Wide Nutrition Studies Reviewed in New Publication

Nutrition Abstracts and Reviews, a new periodical dealing comprehensively with the nutrition of man and the lower animals, has just been issued under the direction of the Imperial Agricultural Bureaux Council, the Medical Research Council, and the Reid Library, Aberdeen, Scotland. Its editorial staff, headed by Dr. John Boyd Orr, is assisted by corresponding editors in 29 other countries. The corresponding editors in the United States are Dr. H. C. Sherman, Columbia University, New York City, and Dr. J. R. Mohler, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture.

Nutrition Abstracts and Reviews contains editorial comments and brief articles by leading authorities, but consists principally of abstracts, classified in six main sections, for the convenience of workers in various fields. The abstracts present scientific findings reported in about 450 periodicals. A short book-review section concludes the volume, the first of which contains 351 pages and covers the period January 1 to June 30, 1931.

BACTERIOLOGY OF SKIN LESIONS OF TUBERCULIN-REACTING CATTLE. Lyman Daines and Harold Austin. Proc. Soc. Exp. Biol. and Med., xxix (1931), 1, p. 3.

More than 90 per cent of the lesions studied came from animals which had given a positive tuberculin reaction. The remainder were not tested. Only one of the 189 skin lesions yielded a typical *Mycobacterium tuberculosis* (bovine type) and this animal apparently had general tuberculosis. Careful search of these skin lesions from tuberculin-reacting cattle revealed the presence in all of them of acid-fast rods, as well as one or more forms of acid-fast or non-acid-fast coccoid, diphtheroid, or streptococcoid organisms or branching filaments. The authors interpret these as forms of a pleomorphic organism. Organs from no-lesion reactors also yielded such cultures. It is possible that this organism is a new, undescribed species or pleomorphic forms in the cycle of a true, attenuated, mammalian tubercle bacillus. Because of their constant occurrence in skin lesions of tuberculin-reacting cattle and in organs of no-lesion reactors, they are regarded as the cause of the skin lesion and of tuberculin hypersenitiveness of most of the reactors in Utah.

A STUDY OF MORTALITY AND EGG PRODUCTION IN FLOCKS AFFECTED WITH INFECTIOUS LARYNGOTRACHEITIS. W. R. Hinshaw, E. E. Jones and H. W. Graybill. Poultry Science, x (1931), 7, p. 375.

The data include studies on mortality and egg-production in 25 outbreaks of infectious laryngotracheitis, affecting 14,574 chickens in producing flocks, in eight poultry districts in California. The average course of the disease in investigated cases was 15 days, with extremes of 7 to 21 days; 85 per cent of all deaths occurring by the end of 15 days, and 98 per cent by the end of the 21st day. The mortality was 1916 birds (13.15 per cent), while the estimated loss in egg-production was 1883 2/3

dozen eggs. The greatest mortality was on the 11th day, and 70 per cent of all losses were from the 5th to the 15th days. The production started to drop 4 days after the onset and reached the pre-disease percentage production 30 days later, with a maximum drop of 12 per cent. The minimum egg-production in the composite group was on the 18th day after the onset of the disease and 7 days after the maximum mortality. The monetary losses from mortality and drop in egg-production were estimated at $2500 and $678.12, respectively, or a total of approximately $3200.

THE THERAPEUTIC USE OF BACTERIOPHAGE AND ITS PRACTICAL DIFFICULTIES. Frank B. Lynch, Jr. Amer. Jour. Clin. Path., i (1931), 6, p. 449.

Several factors are responsible for the variability in the therapeutic effect of bacteriophage. Of primary importance is the accessibility of the pathological focus to the bacteriophage filtrate. If the bacteriophage does not completely destroy the culture, the organisms that survive give rise to a resistant strain of organisms which may still be pathogenic. Bacteriophage is not very effective against blood-borne infections because of the adsorption of the phage by the colloids of the serum. In certain experimental infections, as in peritonitis, in order to get the effect of the bacteriophage it must be injected before or within a half-hour after the bacterial inoculation. The protein in a bacteriophage filtrate has an important bearing on the dosage and choice of method for administration. Bacteriophage may be administered by mouth, irrigation, wet dressing or inoculation. The author stresses the necessity for the standardization of bacteriophage filtrates.

THE EFFECT OF TESTICLE EXTRACT ON ROUS SARCOMA. Donald C. Hoffman, Frederic Parker, Jr., and Thomas T. Walker. Amer. Jour. Path., vii (1931), 5, p. 523.

Experiments are described in which it was found that rabbit testicle extract enhances quite markedly the growth of the Rous sarcoma (I) in chickens following its injection together with the tumor-producing agent. This effect occurs equally well following inoculation of cell-free filtrate of the tumor or with injection of a mash consisting of tumor cells in suspension, the resultant tumors appearing much more quickly in the latter case. Rooster testicle extract prepared under the same conditions as rabbit testicle

extract caused no enhancement. Normal rabbit serum caused but a slight enhancement. The authors do not attempt to answer the question whether this tumor behaves like a true tumor in the sense of mammalian tumors, or whether it is the manifestation of a virus disease.

THYMUS EXTIRPATION IN THE LAYING HEN. Alan W. Greenwood and J. S. S. Blyth. Proc. Soc. Exp. Biol. and Med., xxix (1931), 1, p. 38.

The removal of the thymus gland in the hen led to neither the lowering of the calcium content of the blood nor the production of eggs deficient in shell or egg membrane. Nine immature thymectomized fowls were found to produce eggs with wholly normal shells.

TINCTORIAL TRANSMUTATION OF ACID-FAST MICROORGANISMS AND VIRULENCE OF TUBERCLE BACILLI. Frederick Eberson and Marion A. Sweeney. Jour. Inf. Dis., xlix (1931), 4, p. 303.

Certain environmental conditions, such as cultivation in non-protein mediums, favored the loss of acid-fastness of a strain of tubercle bacilli. Such a strain was avirulent and devoid of invasiveness in guinea pigs. The avirulent, acid-sensitive strain of tubercle bacilli was not allergenic. The organisms disappeared from the site of injection and could not be recovered from the lymph-nodes and organs. Inoculations of suspensions of such material after varying intervals was likewise ineffective. Inoculation of guinea pigs with the avirulent, acid-sensitive organisms failed to protect the animals against reinoculation with a small dose of a virulent culture. The addition of lipoidal or fatty substances to culture mediums did not transform ordinary non-acid-fast bacteria into acid-fast types. This environment was also without specific action in altering the tinctorial characteristics of tubercle bacilli. Virulent, acid-fast bacilli differed strikingly from avirulent, acid-sensitive strains in their reaction to dyestuffs, such as acid or basic fuchsin, neutral fuchsin and methylene blue, that constitute specific reagents in the Ziehl-Neelson acid-fast staining technic. In the avirulent strain, neutral fuchsin favored development of acid-sensitive organisms in an indefinite number of transplants to the same culture mediums. With the other dyes the selective tinctorial effects seemed to be related inversely to the bactericidal action of given dilutions. Acid-fastness was pre-

eminently a characteristic of organisms that resisted the lethal action of concentrated solutions of the given dyes. These studies appear to demonstrate the difficulty of altering the original staining and biologic characteristics of a virulent strain of tubercle bacilli. Similarly, a culture that had become relatively acid-sensitive could not be readily converted into the original acid-fast type. The possibility of dissociating tubercle bacilli by means of certain specific dyestuffs might be inferred from these observations.

Microbic Dissociation in the Brucella Group. M. S. Marshall and Dorothy Jared. Jour. Inf. Dis., xlix (1931), 4, p. 318.

Prolonged cultivation on agar containing specific anti-serum, and repeated selection produces "rough" types of most, probably all, strains of Brucella. The induced "rough" forms are relatively stable. The shift, "smooth" to "rough," appears continuous rather than discontinuous. Differences in metabolism between rough and smooth types probably exist. The R type may be more resistant to the bacteriostatic action of dyes. No correlation was noted in the production of hydrogen sulphid. Only bovine strains of R types may fail to show utilization of dextrose. Guinea pigs were regularly infected by the injection of S types of various origins, with typical pathological changes, low titres of agglutination and positive cultures. Corresponding R types induced few signs of infection. Tests of toxicity in mice failed, a single series being used. An approach to the problem of undulant fever based on the conception of possible and demonstrable variations of strains of any source seems, under present circumstances, to be more logical than an approach based on a conception of relative stability of caprine, bovine, porcine and human strains.

Mucus Secretion in Acute Experimental Inflammation of the Colon, and other Mucous Membranes of the cat. Histological Changes. H. Florey and R. A. Webb. British Jour. Exp. Path., xii (1931), 5, p. 286.

The authors have studied the expulsion of the mucin content of goblet cells in the mucosa of the cat's colon. Under continued irritation after a goblet cell has lost its mucin, a reduction in cytoplasm occurs. There is no sign of desquamation of these cells or suggestion of death in nuclear appearances. The cells at the

bases of the crypts undergo discharge and exhibit their cycle of changes before those nearer the surface. Surrounding a wound, goblet cells did not suffer diminution of mucin content, indicating that products of tissue injury calling forth inflammation do not affect these cells. Mucus-secreting cells of the stomach are much more resistant in their reaction to stimuli than those of the colon. No mucin was found in dividing cells. The reformation of the goblets in the colonic mucosa takes place with considerable rapidity. New cells originate primarily in the lower one-third of the crypt. The authors describe in detail the histological changes taking place in mucus secretion.

Intestinal Carriers of Clostridium Tetani and Immunity. Tetanus IX. George Coleman. Amer. Jour. Hyg., xiv (1931), 3, p. 515.

Tetanus spores fed to guinea pigs over a considerable period fail to become established in the digestive tract after six months. Neither antitoxins nor agglutinins for the type fed were produced in the serum of animals. The serum of these animals showed no prophylactic value against tetanus spores when injected into other guinea pigs. The animals so fed were more immune to the intramuscular injection of spores of the type fed than were normal controls. The author discusses the question of possible immunity to tetanus infection induced by the carrier state.

A Study of Immunization with Bacterial Bodies Against Tetanus. Tetanus VIII. George Coleman and Janet B. Gunnison. Amer. Jour. Hyg., xiv (1931), 3, p. 526.

The authors fail to confirm the report of investigators in China that antispore and antibacterial serum "practically free from antitoxin," which have type specific protective value against infection with *Clostridium tetani*, may be produced in rabbits. By no method employed was any serum produced by bacteria or spores in rabbits, goats, or guinea pigs which contained antibacterial or antispore substances or antibodies effective in preventing tetanus infection in guinea pigs. None of these serums enhanced the protective value of purely antitoxic serums. Antitoxic serums produced with the organisms of *Cl. tetani* as well as with toxoid are no more effective in preventing or delaying infection than those produced with filtered toxoid or toxin alone, the unitage in each case being approximately the same.

QUANTITATIVE AND STATISTICAL ANALYSIS OF INFECTIONS WITH EIMERIA TENELLA IN THE CHICKEN. Frederic Fish. Amer. Jour. Hyg., xiv (1931), 3, p. 560.

Varying infecting doses, from 7 to 630 sporulated oöcysts each, were administered to the hosts and the differential effect of the resulting infections observed. The prepatent period for *Eimeria tenella* infections is 160 to 165 hours, regardless of the size of the infecting dose. An increase in the size of the infecting dose has a directly proportional effect on the resulting asexual infection and on the clinical symptoms of the disease. An increase in the infecting dose has a noticeably direct influence on the total number of oöcysts passed by the host during the resultant patent period. The size of the infecting dose has no effect on the daily oöcyst production; the height of the peak of the resulting infection, nor upon the duration of the patent period. One cannot diagnose species of coccidia from any one sample by size alone. Size is an unreliable specific criterion. Marked changes were noted not only in oöcysts from different hosts harboring the same species of coccidium, but within a single infection in any one host. In general the oöcysts tend to become longer and broader as the infection progresses, the ratio between the two dimensions remaining fairly constant. The volume was found to be more than double during the patent period of the same infection. Shape index, indicating shape without regard to size, is a contributing diagnostic factor for specific differentiation of oöcysts within the host.

THE OCCURRENCE OF BACT. TULARENSE IN THE EASTERN WOOD TICK, DERMACENTOR VARIABILIS. R. G. Green. Amer. Jour. Hyg., xiv (1931), 3, p. 600.

Bact. tularense has been isolated directly from the eastern wood tick, *Dermacentor variabilis*, by guinea-pig inoculation and subsequent culture. Over an area of twenty square miles in central Minnesota, the percentage of wood-tick infection has been found to be less than 0.1 per cent during the summer of 1930. The animal culture derived from ticks was of low virulence, as indicated by the type of lesion produced and by failure consistently to produce fatal infections in rabbits. It indicates that changes in the virulence of the organism occur in nature and may enter as a fundamental factor in the rise and fall of rabbit populations. No increase in virulence was evident from twenty generations passed through guinea pigs by subcutaneous injection or through twelve generations by skin inoculation.

STUDIES OF HOST-PARASITE RELATIONSHIPS OF DIROFILARIA IMMITIS (Leidy) AND ITS CULICINE INTERMEDIATE HOSTS. Stephen M. K. Hir. Amer. Jour. Hyg., xiv (1931), 3, p. 614.

The development of *Dirofilaria immitis* (Leidy) to the infective stage was traced for the first time in five species of mosquitos, *Aedes canadensis* Theobald, *Aedes sollicitans* Walker, *Aedes taeniorhynchus* Wiedemann, *Culex territans* Walker, and *Anopheles punctipennis* Say. In *Culex salinarius* Coquillett, the development of *Dirofilaria immitis* was traced to the intermediate sausage stage of development. Three species of mosquitos, *Aedes vexans* Meigen, *Aedes aegypti* Linnaeus, and *Culex pipiens* Linnaeus, reported by other workers as carriers of this parasite, were also found to be able to serve experimentally as intermediate hosts for *Dirofilaria immitis* in Maryland. There is a marked variation in the range of host susceptibility to infection with *Dirofilaria immitis* in the species of mosquitos tested, both in the average percentage of individuals which become infected in different species and in the intensity of infection as measured by the number of filariae which were able to establish themselves in the host. This nematode is a parasite of the circulatory system of the dog. It is viviparous, the minute embryos being taken in by mosquitos when they suck blood. Here they reach the infective stage and again enter the dog through the skin when the mosquito bites.

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Methods of Segregation for Control and Eradication of Bovine Contagious Abortion. W. B. C. Danks. (Bul. 11, 1931. Dept. of Agr., Colony and Protectorate of Kenya, Nairobi, Kenya.) pp. 16.

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Veterinary Aspects of Undulant Fever. J. F. DeVine. Reprint from *N. Y. State Jour. Med.*, Oct. 15, 1931.

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A Survey of Infectious Laryngotracheitis of Fowls. W. R. Hinshaw. (Bul. 520, 1931. Agr. Exp. Sta., Univ. of Calif., Berkeley, Calif.) pp. 36.

A Study of Mortality and Egg Production in Flocks Affected with Infectious Laryngotracheitis. W. R. Hinshaw, E. E. Jones, and H. W. Graybill. Reprint from *Poultry Sci.*, x (1931), 7, pp. 375-382.

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National Research Council Organization and Members 1931-1932. Washington, D. C., 1931. pp. 62.

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Swine Fever Outbreak of 1927-1928 in New South Wales, The. Max Henry, H. R. Seddon, and C. Blumer. Dept. of Agr., New South Wales. Sydney, Australia, 1931. pp. 138.

PERSONALS

Dr. J. F. DeVine (Amer. '98), of Goshen, N. Y., was scheduled to start on a Mediterranean cruise, on January 26. Mrs. DeVine was to accompany him and they expected to visit nineteen countries in northern Africa, western Asia and southern Europe. They expected to be gone about two months.

Dr. V. H. Miller (O. S. U. '24), of Ottawa, Ohio, had a leg broken in an automobile wreck on the Dixie Highway, between Wapakoneta and Lima, Ohio, early on the morning of December 21. The car in which Dr. Miller was riding crashed into a stalled truck which was parked in the road with no lights showing.

COMMUNICATIONS

INTERNATIONAL VETERINARY CONGRESS REPORTS

TO THE EDITOR:

In view of the forthcoming International Veterinary Congress to be held in Philadelphia in 1934, I should be glad if you would allow me to call the attention of your readers to the fact that a limited number of copies of the reports of the two previous congresses, held in London, are available and can be obtained on application direct to this office.

The three volumes of the report of the Tenth Congress, held in 1914, contain 1750 pages, and the cost is one pound. The three volumes of the report of the Eleventh Congress, held in 1930, contain 1790 pages, and are sold at two pounds. Either or both sets will be sent postfree to any address on receipt of the appropriate remittance, which should be made payable to the Royal College of Veterinary Surgeons.

FRED BULLOCK, *General Secretary,*
Eleventh International Veterinary Congress.

London, England, January 12, 1932.

VETERINARY RESERVE CORPS

TO THE EDITOR:

On page 564 of the October, 1932, issue of the JOURNAL an article appears relative to reserve veterinary officers in the U. S. Army, in which it is stated that the organized Veterinary Reserve Corps has 20 lieutenant colonels, 70 majors, 129 captains, 97 first lieutenants and 257 second lieutenants. I note in the annual report of the Surgeon General, U. S. Army, for 1931, that the following grades and numbers exist in the Veterinary Reserve Corps: Colonels, 2; lieutenant colonels, 24; majors, 97; captains, 286; first lieutenants, 159; and second lieutenants, 410.

The total number of reserve officers (veterinary) given in the article referred to was 573. At present there are 978, an increase of 405 officers or 70 per cent.

The increase in reserve veterinary officers in the Army must be very encouraging to the military establishment and shows that the profession is taking a much needed interest in the national defense.

J. P. TURNER.

Washington, D. C., December 24, 1931.

Regular Army

Major John W. Miner is relieved from further assignment and duty at the remount purchasing and breeding headquarters, Kansas City, Mo., effective on or about January 1, 1932, and will proceed to Fort Riley, Kans., for duty

Major Frank C. Hershberger is relieved from duty at Fort Riley, Kans., effective on or about January 1, 1932, and will proceed to Fort Sill, Okla., and report to the commanding general for duty.

Colonel William P. Hill, Letterman General Hospital, Presidio of San Francisco, Calif., has been directed to report to the president of an Army retiring board at the headquarters, Ninth Corps Area, for examination by the board.

Veterinary Reserve Corps

New Acceptances

Armstrong, Walter Nichol...Capt....Box 3, Concord, Michigan.
Burkey, Fred Morrison.....2nd Lt...1711 Preston Ave., Houston, Tex.

Promotion

Bishop, Wm. Jennings....To: Capt..City Hall, Bartlesville, Okla.

Separation

Casler, Harland Donald.....Major...Died July 11, 1931.

Changes of Address

Cellar, Paul Market.....2nd Lt...350 Orange St., Ashland, Ohio
Newlin, Theo. A.....2nd Lt...521 Convery Pl., Perth Amboy, N. J.
Raebone, Alexander Louden.2nd Lt...Post Box No. 1, Antigua, British West Indies
Wilkinson, Raymond James..1st Lt...Wilkinson Vet. Hospital., 132 Nassau Ave., Kenmore, N. Y.

Tolerances and Analytical Methods Recommended

Recommendations for tolerances and analytical methods for a number of tablets and ampuls appear in the ninth report of the Combined Contact Committee of the American Drug Manufacturers Association and the American Pharmaceutical Association, recently submitted to the federal Food and Drug Administration. The report also contains recommendations relating to the quantity of contents of ampuls.

Free copies may be obtained by writing the Food and Drug Administration, U. S. Department of Agriculture, Washington, D. C.

Honor Society Installs Chapter at Iowa State College

Installation exercises were held on the evening of December 5, 1931, for a new chapter of the Society of Phi Zeta at Iowa State College, Ames, Iowa. The installing officer, Dr. William A. Hagan, Professor of Pathology and Bacteriology at the New York State Veterinary College at Cornell University, and National Secretary of the Society, formally presented a charter to a group of faculty members and students of the Division of Veterinary Medicine, creating the Gamma Chapter of the Society. The following are the charter members of the new chapter:

From the Faculty	*From the Senior Class*
Dr. E. A. Benbrook	Mr. Frank A. Anderson
Dr. H. D. Bergman	Mr. Elwyn W. Coon
Dr. C. H. Covault	Mr. Thos. A. Dermody
Dr. Geo. R. Fowler	Mr. Paul R. Granholm
Dr. H. L. Foust	Mr. Maurice J. Johnson
Dr. Chas. Murray	Mr. Clarence H. Pals
Dean C. H. Stange	Mr. Herbert M. Tabbut
Dr. F. E. Walsh	Mr. Harold E. Wicker
	Mr. Telford W. Workman

The exercises were conducted in the new Student Union Building. Following the installation, there was a dinner in one of the dining-rooms of the Hall, at which brief addresses were given by Dean Stange, Dr. Bergman, Dr. Hagan and Mr. Workman. After the dinner a smoker was held, to which the entire staff of the Veterinary Division had been invited. Dean Stange was elected the first President of the Chapter, Mr. T. W. Workman was elected Vice-President, and Dr. Geo. R. Fowler, Secretary-Treasurer.

The Society of Phi Zeta was founded at Cornell University in 1925, for the purpose of recognizing scholarship and ability among students of veterinary medicine. The Society stands for the highest ideals of conduct and accomplishment. In 1929, the organization was made national in scope and a second chapter was formed at the School of Veterinary Medicine, University of Pennsylvania. The Iowa chapter is the third formed.

The officers of the National Society at the present time are: President, Dr. R. S. Amadon, School of Veterinary Medicine, University of Pennsylvania; Vice-President, Dr. R. R. Birch, New York State Veterinary College, Cornell University; Secretary-Treasurer, Dr. W. A. Hagan, N. Y. State Veterinary College.

W. A. H.

Ohio State Veterinary Conference

The seventh annual conference of the College of Veterinary Medicine, Ohio State University, will be held March 23, 24 and 25, 1932. This year, as formerly, the program is quite an extensive one and includes some of the foremost veterinarians and educators in this country. The entire three-day period will be devoted to the discussion of topics of vital importance to every veterinarian who can be present. A special effort has been made in arranging the details of the program so that its quality and variety will appeal to all veterinarians.

The program this year has been formulated for the purpose of giving to the practitioner the very latest developments in veterinary medicine. A clinical program will be included which should prove to be an added attraction to all interested in the practical side of veterinary medicine. This part of the short course will be for the purpose of definitely illustrating a number of the important surgical and medical problems confronting the practitioner at the present time.

It is generally conceded that such intensive short courses provide the necessary educational opportunities for the busy practitioner. Dean Brumley and his staff hope it will be possible for a large number to avail themselves of this opportunity to receive the very latest information concerning many of the real problems in their various fields of activity.

These short courses are sponsored by the College of Veterinary Medicine believing it can render a real service to the live stock industry through the veterinary profession. In the past years the attendance has been very gratifying to those who have been interested in promulgating these courses. The interest manifested in the programs and the various discussions entered into by those in attendance has been a source of much gratification also. An invitation is hereby extended to all veterinarians to be present on this occasion to receive, we feel sure, much of value from an educational point of view.

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-fourth annual meeting of the Nebraska State Veterinary Medical Association was held at the Elks Club, Omaha, December 8-9, 1931, with approximately 125 members and guests in attendance.

The Association was welcomed to Omaha by Mr. W. L. Pierpont, representing the Omaha Chamber of Commerce. The response was made by Dr. E. J. Jelden, Columbus.

The minutes of the previous meeting were read and approved. The Treasurer's report was then submitted and, same having been audited by a committee composed of Drs. C. C. Hall, D. E. Trump and J. E. Weinman, was approved as read. The presidential address was delivered by Dr. D. E. Trump. The events of the past year were briefly reviewed and recommendations for the future outlined.

The first paper on the literary program, entitled, "The Pathological Anatomy of Anthrax in Swine," was a translation from the Czecho-Slovakian, by Dr. Frank Jelen, B. A. I. Inspector-in-Charge, Omaha, Nebraska. There being very little American literature on this subject, the paper aroused a great deal of interest and numerous requests for copies of same were received.

A report by the Committee on Affiliation with the American Veterinary Medical Association was made by Dr. D. W. Hurst, Chairman, and referred to the Executive Board and Resolutions Committee for action.

The second paper was presented by Dr. A. F. Schalk, Ohio State University, Columbus, Ohio, and was an illustrated lecture on "The Physiology of the Ruminant Stomach." Dr. Schalk had been invited to appear on the 1930 program but was unable to be present because of illness and the Association was doubly grateful for his presence and contribution on this program.

Following lunch, Mr. Frank Lee, representing Swift and Company, of Omaha, offered a display of "Identifiable, U. S.-Inspected, Packaged, Hard-Chilled Meat Cuts," accompanied by a suitable lecture. This was immediately followed by a meat-cutting demonstration by Mr. W. J. Loeffel, of the Animal Husbandry Department, Nebraska College of Agriculture. The ladies were invited to be present for both features, which proved highly interesting to all.

Dr. D. H. Udall, Cornell University, Ithaca, New York, lectured on the subject of "Mastitis." Several specimens were available for reference and, as indicated by the lively discussion which followed, the subject was of timely interest and ably handled.

"The Present Status of Ovarian Hormones," an illustrated lecture by Dr. R. G. Gustafson, University of Denver, Denver, Colorado, was a very unusual feature on the program. Such phenomena as the bringing about of estrum and the development of lactation in the ovariectomized bitch; the changing of color characteristics from male to female and vice versa, in the plumage of birds, by the injection of certain hormones, was ample evidence of the research carried on by Dr. Gustafson and his associates.

The evening was devoted to the annual banquet, which was presided over by Dr. A. T. Kinsley, in the absence of Dr. J. S. Anderson, Dr. R. R. Dykstra, president of the American Veterinary Medical Association, being the featured after-dinner speaker. The Local Arrangement Committee should be congratulated for the splendid entertainment provided and the success of the dance and social evening which followed.

The program Wednesday morning was opened with a report of the Legislative Committee, by Dr. W. T. Spencer, Chairman, in which he outlined the activities of his committee during the year. Dr. A. T. Peters, a visiting veterinarian from Peoria, Illinois, and who served as president of the Nebraska Association in 1896 and subsequently as secretary-treasurer for several years, pointed out the remarkable progress made during the years which have elapsed since the early days of the organization, paying special tribute to Dr. Spencer and his committee. In the course of the remarks which followed, it was stated that the bulletin, "Eradicate Bovine Tuberculosis," compiled by this committee, had been called for by every state in the Union and several foreign countries.

A question-box on "Poultry Diseases," conducted by Dr. Frank Breed, proved to be a very interesting and satisfactory manner in which to handle the subject, as evidenced by the numerous questions submitted and the discussion which followed. Dr. A. T. Kinsley presented a paper on "Infectious Abortion in Cattle." For many years Dr. Kinsley has been one of the featured speakers on our programs and 1931 was no exception.

Dr. Henry Hell, of Wilton Junction, Iowa, president of the Iowa Veterinary Medical Association, presented a paper, "Diseases of Swine." Being a practitioner, the subject was handled in a manner particularly valuable and interesting to the majority of his listeners.

"Facts and Figures," as applied to the Kansas City Milk and Dairy Inspection service, were reviewed by Mr. O. C. Murphy, Milk Commissioner of that city. This talk had been presented at the A. V. M. A. convention in Kansas City and revealed the importance of the veterinarian in this branch of public health service.

Dr. R. R. Dykstra covered the subject of "Equine Practice, with Particular Reference to Surgery of Some of the Head Sinuses in the Equine and the Bovine." This was an illustrated lecture, which helped bring out its practicable application.

Several amendments to the Constitution and By-laws were adopted. Six veterinarians were admitted to membership.

The following officers were elected for the ensuing year: Dr. C. C. Hall, of Omaha, president; Dr. J. S. Barbee, of Sutton, vice-president; Dr. E. C. Jones, of Grand Island, secretary-treasurer. Drs. J. H. Copenhaver, of Ralston, and M. Campbell, of McCook, were elected to the Executive Board, to fill vacancies caused by retirement of Drs. G. J. Collins and W. T. Spencer.

Invitations were extended by the Chambers of Commerce of Lincoln, Omaha and Grand Island for the 1932 convention, the latter being the unanimous choice.

There being no further business, meeting was adjourned.

E. C. Jones, *Secretary-Treasurer.*

NEW MEXICO VETERINARY MEDICAL ASSOCIATION

The first annual meeting of the New Mexico Veterinary Medical Association was held at the Franciscan Hotel, Albuquerque, December 11-12, 1931. Fifteen members and sixteen

visitors registered at the informal gathering on the morning of December 11.

The afternoon session was called to order by Dr. T. I. Means, of Santa Fe, who introduced a number of the visitors to the members of the Association. Following the adoption of the minutes of previous meetings, President Means made some announcements concerning the program, and appointed committees to take care of various matters in connection with the meeting. Drs. Carl E. Freeman and Edward P. Johnson were elected to membership.

Dr. Edward L. Stam, state veterinarian of Arizona, and president of the Arizona Veterinary Medical Association; Dr. E. A. Jennings, of El Paso, Texas, and Dr. A. G. Fisk, of Denver, Colo., were elected to honorary membership.

The following program was presented:

"Coöperation Among Veterinarians—Federal with Others," by Dr. S. W. Wiest, Santa Fe.
"Coöperative Basis of Practicing Veterinarians," by Dr. Carl E. Freeman, Carrizozo.
"Legitimate Veterinary Supply Houses," by Dr. A. G. Fisk, Denver, Colo.
"Coöperation of State Veterinary Departments," by Dr. Edward L. Stam, Phoenix, Arizona.
"Wholesale Drug Supply as It Pertains to Veterinarians," by Dr. E. A. Jennings, El Paso, Texas.
"Tuberculin Testing," by Dr. E. J. Foreman, Trinidad, Colo.
"Matters Concerning All of Us," by Dr. F. L. Schneider, Albuquerque.
"Operation of the Present State Veterinary Law," by Dr. M. C. Wiley, Albuquerque.
"Public Benefits of the New Mexico Veterinary Practice Act," by Dr. C. E. Ackerman, Clovis.
"Further Legislative Work," by Dr. F. H. Barr, Albuquerque.

The annual banquet was held in the Blue Room of the Franciscan Hotel, in the evening, with twenty-five members and guests present. Dr. Carl E. Freeman acted as toastmaster.

At the Saturday morning session the Association was welcomed to Albuquerque by Mr. Clyde Tingley, Mayor. Dr. C. E. Freeman responded. Dr. T. I. Means then delivered his presidential address, after which the following program was presented:

"The Relationship of the Extension Service to the Veterinary Profession," by Prof. W. L. Elser, Director of Extension, New Mexico College of Agriculture and Mechanic Arts, State College.
"Poisonous Plants as a Factor in Live Stock Losses," by Dr. H. E. Kemper, U. S. Bureau of Animal Industry, Albuquerque.
"The Relationship of the Veterinary to the Medical Service in the Army of the United States," by Maj. J. D. Lamon, M. C., New Mexico National Guard, Albuquerque.

Dr. Edward L. Stam then favored the meeting with an address in his usual impressive manner. He stressed higher veterinary

education, preparedness in meeting veterinary problems, co-operation and close contact with the live stock industry upon which our profession depends for its very existence.

At the final session in the afternoon, Miss Myrtle Greenfield, Chief of Division, State Public Health Laboratory, University of New Mexico, ably discussed the subject of the handling of pathological specimens intended for laboratory diagnosis. Dr. H. C. Schipman, of Las Cruces, then presented a masterful paper on the subject of "Sterility."

Following the presentation of several committee reports and the adoption of a number of resolutions, officers for the ensuing year were elected as follows: President, Dr. H. C. Schipman, Las Cruces; Vice-President, Dr. H. E. Kemper, Albuquerque. Immediately following his election, President Schipman announced the following appointments to the Executive Board: Drs. M. C. Wiley, C. E. Freeman, T. I. Means. These three members, with the officers, will constitute the Board for 1932.

F. H. Barr, *Secretary-Treasurer.*

OHIO STATE VETERINARY MEDICAL ASSOCIATION

The forty-ninth annual meeting of the Ohio State Veterinary Medical Association was held at the Neil House, Columbus, January 6-7, 1932. This meeting was most successful from the standpoints of program and attendance.

The first session was devoted to a discussion of problems connected with the use of anti-hog cholera serum and virus. Considerable discussion was developed as to the site of injection, the treatment of pregnant sows, and the care and feeding before and after treatment.

The second session was devoted entirely to the business of the Association. The address of President Zimmer was one of the best ever presented to the Association. It pointed out numerous ways in which the profession could be bettered and suggested many remedies for existing conditions.

The third session was given over to a discussion of bovine mastitis. The use of the colorimetric test was discussed and demonstrated by Dr. E. C. O'Dell, City Health Department, Columbus, and by Dr. J. C. Wickham, City Health Department, Cleveland. Following this, a paper on the care and management of a herd affected with mastitis was given by Dr. C. W. Eddy, Cleveland, and discussed by Dr. C. H. Case, Akron.

At the final session, a very fine discussion on heat stroke in horses was given by Dr. L. J. Richards, of Delaware. This was followed by a lengthy discussion on frights disease and distemper, by Dr. H. E. Myers, Cleveland, and Dr. J. W. Jackman, Columbus. The last subject on the program was "Swine Erysipelas," given by Dr. A. J. DeFosset, Columbus.

The social program consisted of a dinner-dance at the Neil House, for members and their wives, and a complimentary theatre party and luncheon for the ladies.

Officers elected for the ensuing year were: President, Dr. J. N. Shoemaker, Columbus; vice-president, Dr. O. C. Alspach, Marion; treasurer, Dr. D. C. Hyde, Columbus; secretary, Dr. R. E. Rebrassier, Columbus; Executive Committee, Dr. W. F. Guard, Columbus; Dr. H. A. Salt, New Philadelphia; and Dr. Neil H. Myers, Wilmington.

R. E. REBRASSIER, *Secretary.*

ARKANSAS VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Arkansas Veterinary Medical Association was held at Little Rock, January 19, 1932. Dr. W. L. Bleecker, of Fayetteville, presided. The following program was presented:

"Canine Distemper," by Dr. C. E. Salsbery, Kansas City, Mo.
"Buffalo Gnats," by Dr. H. J. Hayes, Helena.
"Progressiveness of Milk Control Work," by Dr. T. M. Dick, Health Department, Little Rock.
"Vitamins," by Dr. Frank Vinsonhaler, Dean, Arkansas Medical College, Little Rock.
"Progress of Area Tuberculin Testing," by Dr. W. A. McDonald, U. S. Bureau of Animal Industry, Little Rock.
"Vitamin and Mineral Deficiency," Dr. C. H. Larsen, Clarksville.
"Live Stock in Arkansas," by Mr. Lee Cazort, Former Lieutenant-Governor of Arkansas.

The following officers were elected for the ensuing year: President, Dr. R. W. Williams, Eldorado; vice-president, Dr. H. J. Hayes, Helena; secretary-treasurer, Dr. Rease Mitcham, Little Rock.

REASE MITCHAM, *Secretary-Treasurer.*

Butter Seized

During the year ended October 31, 1931, food inspectors of the U. S. Food and Drug Administration seized 123 lots of illegal butter. In most instances the seizure actions were based on shortage in butter fat.

NECROLOGY

HENRY B. AMBLER

Dr. Henry B. Ambler, of Chatham, N. Y., died suddenly at his home, August 6, 1931. Although he had been in poor health for about three years, his death was unexpected. An attack of heart disease was the cause of death.

Born in Potterville, R. I., in 1866, Dr. Ambler removed to Spencertown, N. Y., as a boy of seven. He attended public schools and the American Veterinary College. Following his graduation in 1888 he entered the service of the U. S. Bureau of Animal Industry. After a brief stay in government service he decided to enter private practice at Chatham, N. Y.

Dr. Ambler served as president of the village of Chatham and for thirty-five years was active in Columbia County politics. He was a member of the Republican County Committee and of the Executive Committee of that body. He was president of the Chatham Telephone Company, an unusually successful local public utility. He was a member of the Eastern New York Driving Club, a life member of the Columbia County Agricultural Society and of Hudson Lodge B. P. O. E.

One of the last public enterprises of Dr. Ambler was the donation of a splendid park to the village, where children might play in safety. Plans for the development of this park were interrupted by his death.

Dr. Ambler joined the A. V. M. A. in 1890 and had been a member in good standing for 41 years. He is survived by his widow, his mother, a sister and a brother.

HARRY OTIS CHAPMAN

Dr. H. O. Chapman, of Hampshire, Ill., died December 19, 1931, following an illness of one week, caused by pneumonia.

Born at Five Lakes, Mich., March 3, 1893, Dr. Chapman moved to Garden Prairie, Illinois, with his parents, while quite

young. After attending local schools he entered the McKillip Veterinary College. He had completed three years of study when the institution closed. His course was finished at the Indiana Veterinary College in 1922. He entered practice at Marengo, Ill., where he remained for four years. He then moved to Hampshire and practiced there until his death.

Dr. Chapman was in the Medical Reserve Corps from January 15, 1918, to January 15, 1919. He was a member of the American Legion, the Odd Fellows and the Elks. He is survived by his widow, two daughters, his parents and one brother.

C. W. LEHMAN

Dr. C. W. Lehman, of Flanagan, Ill., died at his home, December 30, 1931. He had been a sufferer from diabetes for several years.

Born in Flanagan, August 31, 1870, Dr. Lehman pioneered the practice of veterinary medicine in central Illinois. He was a registered non-graduate practitioner and enjoyed the confidence and respect of an unusually large clientele. At the same time he was held in high esteem by all graduate veterinarians who knew him.

Dr. Lehman's eldest son, Clifford, a graduate of the Chicago Veterinary College, class of 1917, was associated with him in practice.

J. S. K.

ERNEST LEWIS VOLGENAU

Dr. Ernest L. Volgenau, of Buffalo, N. Y., died at his home, January 2, 1932, after an illness of several months.

Born in Brooklyn, N. Y., October 29, 1873, Dr. Volgenau was educated in the public schools of New York and later at the College of the City of New York. He was graduated from the American Veterinary College in 1893. For about a year he assisted the late Dr. W. Horace Hoskins, in Philadelphia. In 1895 he entered the service of the U. S. Bureau of Animal Industry, and continued until 1906. From 1907 to 1912 he was connected with the New York State Department of Agriculture. On September 27, 1913, he was appointed City Veterinarian of Buffalo, a post he continued to fill until his death.

Dr. Volgenau was a member of the New York State Veterinary Medical Society and was one of the organizers of the Western

New York Veterinary Medical Association. He served as president of the latter organization for three years. He held a commission as Major in the Veterinary Reserve Corps. He was intensely interested in Masonic affairs and served as an officer of many organizations. He is survived by his widow and three sons, one of whom, Robert H., is a veterinarian.

HENRY ALVA SMOTHERS

Dr. Henry A. Smothers, of Mount Carmel, Ill., died at his home suddenly, on January 3, 1932, the result of an attack of heart trouble. He was 56 years of age and had been a resident of Mount Carmel for 25 years, having located there following his graduation from the Chicago Veterinary College in 1907. He is survived by his widow, two sons and a daughter.

JASPER BRETT STILL

Dr. J. B. Still, District Veterinary Inspector, Health of Animals Branch, Canada Department of Agriculture, who passed away at his residence in Winnipeg, Manitoba, on January 12, 1932, was born at Strathroy, Ontario, October 28, 1886, coming to the province of Manitoba with his parents when three years of age. He attended local schools and then entered the Ontario Veterinary College. He was graduated in 1907 and then took a year at the McKillip Veterinary College. Following his graduation from the latter institution in 1908, he entered government service.

Dr. Still's first appointment was to the position of Veterinary Inspector in the Health of Animals Branch, which position he held in the provinces of Manitoba and Saskatchewan. In 1918 he was promoted to the position of District Veterinary Inspector for Manitoba, with headquarters at Winnipeg, a position he held at the time of his death.

Dr. Still joined the A. V. M. A. in 1919. He served as A. V. M. A. Resident Provincial Secretary for the years 1921-22 and 1923-24. He was a member of the Veterinary Association of Manitoba and was serving that body as president at the time of his death. Previously he had held the office of secretary-treasurer and registrar for a number of years.

Surviving Dr. Still are his widow (née Frances Bessie Guy), four children, his mother and three brothers.

W. H.

GEORGE M. REYNOLDS

Dr. George M. Reynolds, of Zionsville, Ind., died in the Methodist Hospital, January 14, 1932.

Born at Georgetown, Ill., October 20, 1883, Dr. Reynolds was educated in the public schools of Illinois and was graduated from the Indiana Veterinary College in 1911. He engaged in general practice at Mooresville, Ind., for two years and then became associated with the Pitman-Moore Company, at the biological farm at Zionsville. He remained with the company until about five years ago, when he accepted a position with the Indiana State Live Stock Sanitary Board, as inspector and quarantine officer.

Dr. Reynolds joined the A. V. M. A. in 1920. He was a member of the Indiana Veterinary Medical Association, a past master of the Zionsville Masonic Lodge and recently was installed as worthy patron of the Order of Eastern Star at Zionsville. He is survived by his widow (née Florence Elmore), his mother, three sons, one daughter and one brother.

WILLIAM H. MURRAY

Dr. William H. Murray, of Memphis, Tenn., aged 62, died in an ambulance on the way to a hospital, November 30, 1931, as the result of a heart attack. He was a registered non-graduate practitioner and had been located in Memphis for 28 years.

GEORGE HENRY THOMAS

Dr. George H. Thomas, of Gibsonburg, Ohio, died in Mercy Hospital, Toledo, Ohio, January 7, 1932, aged 67. He had been ill three months. He was a registered non-graduate practitioner, having attended the Ontario Veterinary College without finishing the course.

L. B. PHELPS

Dr. L. B. Phelps, of Poynette, Wis., a registered non-graduate practitioner, died January 12, 1932, following a stroke. He was 85 years of age and had practiced for 40 years.

Our sympathy goes out to Dr. Alexander Glass, of Philadelphia, Pa., in the death of his wife, Elizabeth Godber Glass, December 31, 1931.

PERSONALS

MARRIAGES

Dr. C. H. Hays (O. S. U. '08), of Pierre, S. Dak., to Miss Hannah Keane, of Saint Joseph, Mo., November 28, 1931, at Saint Joseph, Mo.

Dr. Kenneth L. Benner (O. S. U. '31) to Miss Amelia Patterson, both of Bainbridge, Ohio, December 31, 1931.

BIRTHS

To Dr. and Mrs. Harvey L. Fell, of Wilmington, Del., a daughter, December 4, 1931.

To Dr. and Mrs. K. A. Frisch, of Kenosha, Wis., a son, January 14, 1932.

PERSONALS

Dr. C. L. Campbell (O. S. U. '26) has removed from South Bend, Ind., to Mexico, Mo.

Dr. L. A. Wileden (Mich. '13), of Mason, Mich., is head of the City Welfare Department.

Dr. W. C. Bowen (O. S. U. '31), of North Lewisburg, Ohio, has located at Richwood, Ohio.

Dr. George Scheetz (O. S. U. '28) has removed from South Charleston, Ohio, to London, Ohio.

Dr. R. M. Nyblett (Ont. '02) has left Lonesome Butte, Sask., and is now in Victoria, Brit. Col.

Dr. W. L. Scofield (Gr. Rap. '11), of Athens, Mich., was recently appointed Calhoun County Live Stock Agent.

Dr. E. M. Rogers (Chi. '10), who was formerly located at Rice Lake, Wis., has opened an office in Shell Lake, Wis.

Dr. R. L. Ramsey (Gr. Rap. '06), of Lapeer, Mich., who has been ill for several weeks, is reported to be recuperating slowly.

Dr. S. W. Schuppan (Amer. '90), formerly of Jekyll Island, is now at 809 Monk St., Brunswick, Ga., according to a recent advice.

Dr. L. E. Thompson (Chi. '04), of Newman, Ill., is reported to be planning to move to Alva, Okla., where he will engage in practice.

Dr. Geo. Kernohan (K. S. C. '12), formerly located at the University of California, Berkeley, gives his new address at Draper, Utah.

Dr. M. M. Fletcher (Chi. '06), of Illiopolis, Ill., was bitten by a dog believed to have been suffering with a rabies, the first week in January.

Dr. J. R. Currey (Ont. '29) recently announced the opening of the Washington Animal Hospital, at 4238 Wisconsin Ave., Washington, D. C.

Dr. Frank E. Morgan (Mich. '25), who has been located at Merrill, Mich., has returned to Hubbardston, Mich., to engage in general practice.

Dr. F. Eugene Carroll (K. S. C. '28), formerly a member of the veterinary faculty at Texas A. & M. College, is now located at Fredericktown, Mo.

Dr. C. C. Watts (O. S. U. '14), of Circleville, Ohio, was elected president of the Circleville Chamber of Commerce, for 1932, at the recent election.

Dr. G. B. Merrick (O. S. U. '31), of Shannon, Ill., is reported to have undergone an operation for goiter, in a Cleveland, Ohio, hospital, recently.

Dr. Clarence B. Denman (O. S. U. '09), of Newark, Ohio, has announced his candidacy for the Democratic nomination for sheriff in the spring primaries.

Dr. E. R. Steel (Corn. '14), of Kansas City, Mo., continues to improve. He reports that he is now able to do considerable work and is feeling much better.

Dr. C. McCandless (Chi. '15), of Salem, Ohio, was kicked on the head by a horse, the latter part of December. Dr. McCandless was recovering, according to the latest report.

Dr. John J. Larson (Chi. '18), formerly located at Battle Lake, Minn., has removed to Saint Cloud, Minn., where he is now engaged in general practice. Address: 514 First St. N.

Dr. Chas. R. Schroeder (Wash. '29), of Pearl River, N. Y., recently accepted a position with the Zoological Society of San Diego, Balboa Park, San Diego, Calif., effective February 1.

Dr. S. Holmes (McK. '14) has removed from Marengo, Ill., to Hampshire, Ill. During the late war Dr. Holmes was a captain in the Royal Army Veterinary Corps, British Expeditionary Forces.

Dr. Lloyd D. Jones (Iowa '31), who has been associated in practice with his father, Dr. F. E. Jones (McK. '99), at Rochelle, Ill., has taken over the practice of the late Dr. H. O. Chapman, at Hampshire, Ill.

Dr. I. B. Boughton (O. S. U. '16), following several months of post-graduate work at Ohio State University, recently accepted a position at the Texas Agricultural Experiment Station, Sonora, Texas.

Dr. Frank George (O. S. U. '16), of Plain City, Ohio, was painfully injured when the rim of a school bus tire flew off while he and another man were attempting to remove it before the air had been expelled.

Dr. V. E. Fisher (Iowa '30), who was located at Groton, S. Dak., during the past summer, recently accepted an appointment in the U. S. Bureau of Animal Industry and has been assigned to Newark, N. J.

Dr. C. H. Thomspon (Cin. '10), of New Vienna, Ohio, narrowly escaped more serious injury when a cow he was testing squeezed him against a post. Dr. Thompson was forced to take a rest of about a week to recover.

Dr. F. B. Batten (O. V. C. '94), of Lexington, Ky., was obliged to undergo an operation in a local hospital, in December. A letter dated January 11 stated that he was up and around again and on a fair way to recovery.

Dr. George C. Moody (Ont. '85), of Mason, Mich., has been reappointed Ingham County Veterinarian. Dr. Moody is in his 88th year, which probably makes him one of the oldest, if not the oldest, graduate veterinarian who is actively discharging the duties of the office of county veterinarian.